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Archives of Neurology and Psychiatry

Vol. 4 JULY, 1920 No. 1

THE NEUROSES OF PEACE *

JAMES HENDRIE LLOYD, M.D.
PHILADELPHIA

In retiring from this chair, with thanks for the honor accorded me, I have thought it worth while not so much to review the past as to forecast the future. Nevertheless, the work of the Society for the past year is worthy of mention, for it has been both active and productive, so much so that I make bold to suggest that we might with advantage add one more monthly meeting to our list, in order the better to give vent to our somewhat pent up energies. Our best enterprise was our excursion to Cape May to study the war cases of Dr. Frazier and Dr. Ingham, before which we had had a special program devoted to the military experiences of others of our members.

We have, in fact, had our minds much fixed on the neuroses of war, until, I believe, the time has come for us to turn the page and to begin to think about the neuroses of peace. It is apparent that the dangers of peace may be even greater than the dangers of war, and the panorama of the world—as spread before us today—may make the timid exclaim with King Pyrrhus, "Another such victory, and we are lost." These world phenomena appeal to us not as politicians but as physicians, not as partisans but as scientists; it is for us to discover the pathologic in them, for even in the mad whirl of pandemic hysterias in which so many others are losing their heads, the neurologists may be trusted to keep theirs on their shoulders. Let us gather the fruits of a disastrous victory, for sometimes the gleaning of Ephraim is better than the vintage of Abiezer.

The condition in which the world finds itself imposes an obligation on such a society as ours. It should aim to take a lead in sane thinking; to oppose with a persistent and trenchant criticism the vagaries of thought in high and low places: to stem the turbid tide which bears on its bosom the flotsam and jetsam of a neurasthenic age.

A recent writer in *Harper's Magazine* exults in the thought that this age and country is that of the commonplace. In his opinion nothing above a low average level is to be, or should be, allowed to survive.

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* Presidential Address before the Philadelphia Neurological Society, Jan. 23, 1920.

This writer represents the class consciousness of the mob. His millennium would be a sort of monkey paradise; but his ideas are, of course, in direct opposition to all the teachings not only of history but also of biologic science. If anything has been proved it is that progress takes place by conflict and the survival of the fittest. He and other Philistines like him are the children of the devil, not for any strength they have in them, for they have no strength; but for their very weakness, their inanity, their neurasthenic outlook and example. Many of them will procreate the specimens of dementia praecox who will form the clinical material for a future generation of Philadelphia neurologists.

REACTIONS OF WAR

For what do we see in Europe today? Old empires which for centuries had held Europe together in the bonds of some sort of civilization, are shattered into bits; and in their places is a saturnalia of riot under the aegis of certain light-hearted political philosophers who proclaim that there must be a self-determination of small peoples. These small peoples proceed to cut each others throats, determined only that nothing shall survive above the lowest level of the lowest proletariat. This new freedom has thus brought about a crisis, the ultimate effects of which no man can foresee; but for the neurologists (who are *not* politicians) it means larger hospitals, larger insane asylums, more cranks, more psychasthenics, more political assassins, more medicolegal trials in our criminal courts, more experts and more fees. I am not a pessimist, for I regard pessimism as the cheapest kind of threadbare philosophy, but it is better than the superficial optimism which is the bane of much of our public thinking in America.

Dean Inge, of St. Paul's Cathedral, London, in a recent sermon, has advanced a startling theory. It is no less than this, that western civilization has received a mortal wound in the late war. His idea seems to be that the human race can no longer bear the burden of our hard, mechanical and industrial civilization. He puts it with brutal frankness. The mass of our city population finds industry and life under twentieth century conditions repellant and unendurable, and is going to refuse to continue to produce. Men were not made for this sort of thing. It has grown to be monstrous and clean out of hand. It incarcerates men in factories and mines under abnormal conditions; it herds them in narrow streets and unhygienic dwellings; it gives them unsatisfying wages for a life of industrial slavery. Dean Inge does not say so, but it is a fact that civilized man is not far removed in point of time from the Stone Age. If you scratch him skin deep, you will find the primitive savage. If you will read Lyell's "Antiquity of Man," or

Osborne's "Men of the Old Stone Age," you will be impressed with something like a shock to find that the period of man's civilization, since the earliest records in Egypt and Babylonia, is but as yesterday compared with the long prehistoric period during which the human race existed in Europe as the contemporary of the cave-bear, the cave-hyena and the rhinoceros. Man goes back to the Pliocene period, and it is estimated that 100,000 years will not cover his career. Out of all that squalor, when he wore a piece of skin about his loins, fed on oysters and shell-fish, and fought for his cave with the hyena and the bear, he is called on in a comparatively brief period, without time for adjustment to his new environment, to endure the grievances of a highly complex and relentless civilization. Here arise some profound biologic questions. The history of evolution seems to prove, as you can read in Darwin and Haeckel, that when any species (high or low) is suddenly subjected to a new environment to which it cannot adjust itself, it invariably perishes from the earth. This is the history of the mastodon, the sabre-toothed tiger, the dodo and many other forms. The question arises, Is it to be the history of industrial man? Perhaps the only thing that will save him will be the exhaustion of the coal beds, for our gigantic modern system is founded on coal, and experts predict that the coal will be gone in a century or so. Within our own time the price of it has doubled. Perhaps when it is gone, mankind will have another chance to save itself, and to prove that the greatest advances of the human spirit, in the future as in the past, do not depend on the steam engine and the coal mine.

This thing is going to take a frightful toll of human brains and human nerves. The reaction is inevitable, and we see the signs of it not only in Europe but also in this country. Large portions of the population in America are in revolt. Large portions of the population of Europe are starving. Only this week the Austrian government — such as it is — is offering for sale or rent all the palaces, castles and imperial domains in the hope of attracting American millionaires to take them, and thus provide money to rescue little children from starvation. They are sending abroad pictures of these little ones in the last stages of inanition. A once proud empire thus pays the penalty for a false civilization.

The brutalizing effect of the late war was appalling. The atrocities perpetrated by some of the combatants were unparalleled in human history. The mask was flung off; the cave man returned to his own. But the shock, the scar remains. There is a sort of traumatic neurasthenia that has laid hold, not only of individuals, but of large masses of people.

PSYCHOLOGY OF THE STRIKE

Some one should write a paper on the psychology of the modern strike. One idea acting on large masses of men is its distinguishing feature. It is a manifestation of the instinct of the herd, when stampeded, to go in one direction, without heed to the dangers incurred or the obstacles to be overcome. There is also a dim instinct of self-preservation. It thus becomes an obsession, leading men to clamor for self-aggrandizement while ignorant of economic realities and indifferent to the claims of society at large. When it gathers sufficient momentum, as it can in a vast country like the United States, it becomes formidable by its mere bulk. It is pathologic because it partakes of the nature of a pandemic psychosis. It is a sort of folie communiquée — an example of mental contagion on a large scale. There is an underlying paranoid state, or a sense of persecution, which leads to acts of resentment and violence.

PROHIBITION A PANDEMIC HYSTERIA

But if we want to see a real pandemic hysteria, we have not far to go in this country; and it is one that did not take its origin in war, although the war helped it along wonderfully. Prohibition is the greatest pandemic hysteria since the Crusades, and it is not unlike the Crusades in some respects, for it makes its appeal to ethical and religious enthusiasm. But the Crusades lasted for nearly two centuries, so if there is any truth in the analogy, our bibulous friends need not look for relief at an early date. In fact, these moral movements have sometimes great longevity. Mahomet forbade wine in the Koran twelve hundred years ago, and that prohibition still stands in Mahometan countries, although it encourages the abuse of opium, and it did not prevent Omar Khayyam from singing the praises of the "old familiar juice." Prohibition in America took its origin largely as an industrial necessity; it is a genuine product of our industrial age. It is aimed largely against the abuse of alcohol by our industrial population. Its advocates are never weary of holding up the frightful example of the wage earner who spends his wages on drink and then goes home and beats his wife; and the employer of labor supports it because, as he will tell you, it promotes efficiency. Therefore, if it is good for the working man, it is good for all of us, for we are all brought down to a common level. Neurologists have little reason to defend alcohol, but neither can they view with complacency a popular movement which overwhelms everybody; which makes of us all a holocaust. Prohibition is a demonstration of the failure of the American people in self-government. Our fathers left us a liberal and rational constitution, but time has proved that it was too good for us. We must need patch and disfigure it with a sumptuary law which would do credit to a village constabulary. Last week the newspapers announced that several thousand agents of the Revenue Department were let loose in this city to watch for moonshiners, boot-leggers and the manufacturers of hair tonics and ethereal highballs. These spies have the right to enter our houses, search our cellars and closets, and carry us off to jail if we don't look out. It remains to be seen what the effect of prohibition will be in reducing crime, poverty and insanity. As medical men we are bound to be impartial—but in a former paper I showed by statistics that the rôle of alcohol as a cause of insanity had been greatly exaggerated. Some of the figures were taken from the Pennsylvania Hospital for the Insane, and showed that among the female patients only about 6 or 7 per cent. had been sent there through the abuse of alcohol.

HYSTERIA OF SPIRITUALISM

But even in this world of doubt there is ever some hope. This is the time for us in Philadelphia to receive messages from the unseen world, and I have to apologize to this society for having neglected to secure the services of Sir Oliver Lodge. He and the Reverend Conwell could make us a great neurologic séance. But why are the messages which they receive from the spiritual world always about such trival matters? Does it need a voice from Heaven to tell a man where to find his gold pencil? I should like to question them about more weighty matters. Perhaps the spirits, if properly approached, could resolve some of our doubts in neuropathology. They might tell us, for instance, where the spirochete first gets in his work in locomotor ataxia, or clear up the mysteries of the circulation of the cerebrospinal fluid. Perhaps they could tell us about the functions of the red nucleus, or whether the corpus striatum is really a vestigial organ. But for a great British scientist to come all the way to America to inform our simpleminded people about a lot of old trumpery superstitions, makes me suspect that he is better fitted to be a dancing master of another St. Medard. His advent among us is a psychologic phenomenon. He is a sign of the times. We may yet have a witchcraft craze, right here in town, that will eclipse the historic one in Salem, and give us all an opportunity for first-hand study. I rather wish we would. These perversions of thought, these hysterias, these pandemics are wonderfully interesting if you only take them in the right way; and they would make a good year for this Society.

DECLINING BIRTH RATE

As we cast our eyes over a distracted world, we must not neglect to note the rather alarming decline in the birth rate in some civilized countries, especially in France. France has been losing population from this cause at the rate of several hundred thousand a year, and is threatened, we are told, with extinction. Is the cause merely economic, or is it due to some occult decadence in civilized man? Neurasthenics are not good breeders—the prospect appalls them. Dr. Richet, before the Académie de Médecine in Paris, said that the cause was merely economic, and that France could increase the number of births per year from 700,000 to 2,000,000 by merely offering a premium on babies. The aberrations of thought on this subject are amazing, and quite in accord with the spirit of the times. Thus the French Chamber of Deputies has just rejected by the narrow margin of 219 to 200 votes a bill granting plural ballots according to the number of children, and the Senate was still seriously considering it. By this bill a man would have an additional ballot for every child: thus for three children three ballots; for five children five ballots, and so on. I do not know how this would work in France, but I think I know how it would work in Philadelphia. It would increase the population in the Fourth Ward, but not around Rittenhouse Square. In other words, it would tend to increase the submerged tenth, but not the better elements. This scheme to promote procreation by stuffing ballot boxes, reminds me of something that Carlyle tells us in his "French Revolution." When the terror was at its height, and popular frenzy triumphant, a cry went up (just as now) that France was losing population, whereupon queer fashions arose in women's dress — but you can read about this for yourselves in the third volume of his history. Moreover, this scheme is an attempt to establish a sort of child-suffrage (by proxy); and it may be compared with woman suffrage. It is a curious display of the doctrine that political perfection can be attained by counting noses. It also proves that freak legislation is not peculiar to America. And speaking of the French Revolution may remind us that during and after that cataclysm mental and nervous disorders were much increased.

PROBLEMS FOR THE NEUROLOGIST

Burke said that you cannot indict a whole people — neither, I suppose, can you indict a whole age. My object is merely to make an appraisement of some of the problems of the times, especially as they present themselves to the neurologist. They offer some tempting themes for this Society for the coming year, and I trust they will find a place on our programs. The labor unrest in this country alone is bound to show some psychiatric features. We are not concerned with its political or economic aspects except as these display a pathologic trend. We must also oppose the rampant idealism of the times; we must champion the realities. It is some of these realities, as they consist in shattered nerves and unbalanced minds, as they emerge from the nebulous realms of a false idealism, that will demand our best thought. We are in danger of being swept into some millennium or other before we know it; and I know of no worse fate than to wake up some fine morning and find oneself in a millennium. It would be a sort of league of hallucinations, in which even the most expert neurologist might find it difficult to find his way. But if we are realists, we are not materialists. We do not sympathize with the perversion of esthetics to utilitarianism, with the onslaught on higher education, with the apotheosis of the mob, with the repressed emotions of the freudians, or with the expressed emotions of the parlor bolshevists. If we elect to turn away from the crude radicalism of the day, and to embrace a rational conservatism in medicine and in thought, it does not follow that we are pagans, suckled on a creed outworn.

TRIBUTE TO OSLER

There has just passed from the stage of this world a former illustrious member of this Society, and I would pause a moment in closing to pay a tribute to the memory of Osler. In our time no man has done more than he to promote the general culture of our profession. With a sane mind, a wide outlook, and a fluent pen, he was the natural foe of all philistinism and mediocrity, and the medical world is forever indebted to him in that in a materialistic age he always held up the true ideal of the physician, the scholar and the gentleman.

ELECTROMYOGRAPHIC STUDIES OF MUSCLES DURING HYSTERICAL CONTRACTION *

STANLEY COBB, M.D.

BOSTON

This short communication is written to put forward some laboratory evidence that the involuntary muscular contractions in hysterical conditions are physiologically similar to normal, conscious muscular contractions. From the clinical standpoint their similarity has, of course, been conceded for a long time. In any discussion of hysteria, however, it must be remembered that one is dealing with a complex disease, the pathology of which is psychologic; in other words, this pathology has its seat in the most highly integrated part of the organism. The question in such a piece of work as this is what difference, if any, one might find in the muscular contractions in states of normal psychobiologic integration as compared to the muscular contractions in states of hysterical malintegration.

Action-currents are slight changes of electrical potential concomitant with all forms of biologic activity. A record of the action-currents of a contracting muscle as recorded by the string-galvanometer is therefore termed an electromyogram. The string-galvanometer has been used in the study of the skeletal muscles by Piper,[1] who finds that when a muscle is voluntarily contracted a series of action-currents is recorded at a frequency of about 50 per second as long as the contraction lasts. This takes into consideration only the main waves (Hauptwellen) and not the wavelets which are superimposed on the larger waves. Recently Forbes [2] has criticized and amplified this work. By referring to either of these authors a detailed description of the methods may be obtained. In brief, the technic consists of placing over the muscle to be studied a nonpolarizable electrode which leads off the action-currents of the contracting muscle to the string of the galvanometer, the circuit being completed through another similar electrode on the skin near the first, but not over a contracting muscle. The movements of the galvanometer string are recorded by throwing a beam of light on to this string and photographing its shadow on a moving film. The method is the same as that used for making electro-

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* This paper represents a part of the work done during 1919 as Dalton Scholar at the Massachusetts General Hospital.

[1] Piper: Arch. f. Anat. u. Physiol. (Phys. Abth.), 1914, p. 345. Ibid.: Electrophysiologie Menschlischer Muskeles, Berlin, 1912.

[2] Forbes and Gregg: Am. J. Physiol. **37**:118, 1915. Forbes and Rappleye: Am. J. Physiol. **42**:228, 1917.

cardiograms; in fact, with a few minor changes the same apparatus can be used. The technic used in these cases was the same as that used by Forbes except that in Cases 3 and 6 German silver electrodes were used instead of the nonpolarizable zinc sulphate electrodes. These are more easily applied to uncontrolled muscles, but do not give as sharp detail in the myogram.

The literature on electromyography is small, and there are no published records of spontaneous, hysterical muscular contractions. Gregor and Schilder [3] have shown the effect of fright on muscular contraction in normal subjects. They also have published electromyograms of katatonia. Apart from this, all the clinical work has been on phenomena with a more grossly organic pathology, such as hemiplegia, tabes,[4] ankle and patellar clonus [5] and chorea.[6] Wertheim Salomonson [7] has published two electromyograms of ankle clonus, one "organic" and the other "hysterical."

OBSERVATIONS ON HYSTERICAL MUSCULAR CONTRACTION

Electromyograms were made of six cases which showed hysterical muscular contractions. As is shown in the table, the patient in Case 1 had tonic contractions of the extensor muscle groups of both legs. The patient in Case 2 showed muscular cramps of short duration. The patients in Cases 3 and 4 had still shorter contractions occurring at more or less regular intervals and thus resembling tremors, while the patients in Cases 5 and 6 had typical convulsive tics, one of the sternomastoid and the other of the rectus abdominis. The last ten records are controls, the records being taken from various muscles on six normal persons.

A study of the table and a comparison of the myograms with one another, and with other published records of normal contractions, shows that the hysterical muscular contraction gives an electromyogram similar in all essential points to a voluntary, consciously controlled muscular contraction. In general, the hysterical contractions are less well sustained and less powerful, but in so far as their electrophysiology is concerned the two kinds of muscular contraction are the same.

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[3] Gregor and Schilder: Ztschr. f. d. ges. Neurol. u. Psychiat., Original **18**:195, 1913.

[4] Gregor and Schilder: Ztschr. f. d. ges. Neurol. u. Psychiat., Original **14**:359, 1912-1913.

[5] Gregor and Schilder: München. med. Wchnschr. **59**:2851, 1912. Cobb: Johns Hopkins Hosp. Bull. **29**:247, 1918.

[6] Cobb: Johns Hopkins Hosp. Bull. **30**:35, 1919.

[7] Salomonson: Folia Neurobiol. **4**:1, 1910.

Hysterical Contractions in Author's Cases and Controls

Case Number	Diagnosis	Description of Muscular Movement	Muscle from Which Record Was Taken	Character of Electromyogram	Frequency of Main Waves per Second
1	Hysterical tonic contraction	Both legs were fully extended at knee and ankle by a steady tonic contraction of the extensor muscles that was only relaxed during sleep and that could only be overcome by application of great force. There was partial flexion of the thighs on the trunk and slight adduction of both legs; duration ten weeks. Cured by suggestion	Left tibialis anticus Left rectus femoris	Continuous tetanus of action-currents Same	68 64
2	Hysterical muscular cramps	Every few minutes the patient had a series of muscular contractions beginning with grimacing and torticollis due to contraction of the platysma and sternomastoid, then the trunk stiffened out and rotated to the right and the great toe and foot extended dorsally. The spasms lasted about ten seconds, in any one muscle about four seconds; duration of the disease was four months. The patient improved by suggestion	Left tibialis anticus	Continuous tetanus of action-currents from three to five seconds	60
3	Hysterical tic of arm muscles	Short periodic contractions of the muscles of the right arm occurring about twice per second and lasting 0.3 of a second. The biceps flexed the elbow, and forearm flexors and pronators rotated the hand and flexed the fingers. Duration three months. Helped by suggestion, but relapses under emotional stress	Right biceps	Short periodic groups of action-currents	45
4	Hysterical tremor	When the right arm was extended there was a slow rhythmic rotation movement of the forearm and slight flexion and extension of the third, fourth and fifth fingers, with an increase of the extension at the elbow	Right triceps	Steady tetanus of action-currents with increase at time of movement	50
5	Hysterical tic of neck muscles	Spasmodic contractions of the right sterno-mastoid muscle and platysma occurring every five or ten seconds and pulling the chin downward and toward the left shoulder	Right sterno-mastoid	Short groups of action-currents occurring irregularly	55
6	Hysterical tic of abdominal muscles	Short sharp contractions of both recti abdominium at intervals of about three seconds and lasting about two fifths of a second. These began ten months ago with vomiting and retching. Helped somewhat by suggestion	Left rectus abdominis	Short groups of action-currents occurring irregularly	50
7	Normal control	Voluntary plantar flexion of foot....	Right gastrocnemius	Steady tetanus	50
8	Normal control	Voluntary grip with right hand......	Flexor sublimis digitorum	Steady tetanus	58
9	Normal control	Voluntary grip with right hand......	Flexor sublimis digitorum	Steady tetanus	47
10	Normal control	Voluntary grip with right hand......	Flexor sublimis digitorum	Steady tetanus	48
11	Normal control	Voluntary grip on dynamometer with right hand	Flexor sublimis digitorum	Steady tetanus	52
12	Normal control	Voluntary grip on dynamometer with right hand	Flexor sublimis digitorum	Steady tetanus	63
13	Normal control	Voluntary contraction of biceps by flexing elbow against pressure	Right biceps	Regular steady tetanus	47
14	Normal control	Voluntary contraction of triceps by extending elbow against pressure	Right triceps	Steady sustained tetanus	65
15	Normal control	Voluntary contraction of sterno-mastoid muscle elicited by pressing chin toward left shoulder	Right sterno-mastoid	Steady tetanus with irregularities due to shift of leads	50
16	Normal control	Voluntary contraction of tibialis elicited by pressing upward with foot against a weight	Left tibialis anticus	Steady sustained tetanus	48

The frequency of the main waves[8] varies between 68 and 45 per second, the average being 56, for the hysterical patients. Among the controls the maximum frequency per second is 65 and the minimum 47, with an average of 53. This difference is not significant since the possible error in counting is large. These main waves are counted by taking a place in the record where the action-currents are running regularly and rhythmically, and measuring their duration, thus computing how many would occur per second if this regularity were constant. Where the contraction is well sustained and the series of action-currents clearly defined, the waves may be counted for a whole second. In such cases the averaging and measuring is unnecessary.

In former papers on this subject I have counted the small waves or wavelets as well as the main waves, but experience has shown that the number of these wavelets varies greatly with the technic of the record-making: if the film is run fast, more detail is seen and more wavelets appear; in slower records the wavelets cannot be seen and so are not counted. So too, the resistance of the patient affects the amplitude of the waves—if excessive, the wavelets are obscured.

OBSERVATIONS ON COARSE HYSTERICAL TREMORS

The muscular contractions in Cases 3 and 4 were periodic and thus had some clinical resemblance to coarse tremors; Case 3 might have been mistaken for paralysis agitans or a syphilitic tremor, and Case 4 for the intention tremor of multiple sclerosis. The electromyograms, however, show that the rate of both tremors is much slower and their periodicity is more irregular than the rates and periodicities found in the typical tremors associated with definite lesions of the central nervous system. In Case 3 (Fig. 3) the contractions come at the following periods measured in seconds: 0.55- 0.60- 0.48- 0.40- 0.40- 0.45- 0.50- 0.45- 0.60- 0.63- 0.80. In Case 4 (Fig. 4) the periods are 0.70- 0.60- 0.70- 0.55- 0.80 second. An electromyogram from a case of paralysis agitans is shown for comparison (Fig. 5). This was made from the gastrocnemius of a typical case in which there was a marked unilateral tremor of both arm and leg. The electromyogram shows a remarkably regular periodicity (0.15- 0.17- 0.15- 0.15- 0.15- 0.17- 0.16- 0.17- 0.15- 0.15- 0.15- 0.17- 0.17- 0.17- 0.17- 0.17-) making a tremor rate of about 6.2 per second.

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[8] The difference between the frequency of these main waves of the action-currents, and the rate of tremor must be kept clearly in mind. Action-currents accompany muscular activity; a single muscular twitch may have but one concomitant action-current; short groups of action-currents accompany each muscular contraction of a tremor or a clonus, and these groups appear periodically, and at a rate corresponding to the rate of the tremor. Making up any one of these periodic groups, however, are from two to a score of action-currents which have a wave length and frequency of their own. Thus, in Case 3 (Fig. 3) the rate of tremor is about 2 per second and the frequency of action-currents 45 per second.

Clonus may also be regarded as a form of tremor, although it is not spontaneous and is only set up by stimulation of the proprioceptors. Electromyograms of ankle, patellar and wrist clonus show a typical rate of 6 or 7 per second. One published case[9] with an average rate of 6.6 per second showed one consecutive series of 160 contractions where the periods, from the beginning of one contraction to the beginning of the next, each measured 0.15 second. These electromyograms of clonus are almost identical in periodicity, regularity and rate[10] with those taken from the coarse tremors of paralysis agitans and neurosyphilis. In paralysis agitans, however, many cases show a slight continuous electrical activity of the muscle (Fig. 5.—V, V) even between the tremor contractions (Fig. 5.—C, C) which are accompanied by the large action-currents. This is especially marked in those cases in which the tremor is only present when the muscles are first put into a state of moderate tension either voluntarily or through emotional stimuli. This has also been noted for clonus.[5, 7] Wertheim Salomonson shows an electromyogram from a case of "real clonus" and compares it with the curve given by a case of "hysterical clonus." The former is clean cut and with only one or two action-currents per clonic contraction, and no electrical activity in the muscle between contractions; whereas the "hysterical" case shows a slight constant tetanus of action-currents at a frequency of 50 per second with superimposed on this a series of larger action-currents occurring synchronously with the clonic contractions, as is shown by a simultaneously made mechanical myogram. He concludes from these facts "that a voluntary contraction underlies the clonus that is sometimes found in normal persons or in hysteria patients" and that "this voluntary tetanus constitutes the mechanical condition necessary for the genesis of clonus. The muscular hypertony in cases of lateral sclerosis and allied diseases provides the mechanical condition which renders the occurrence of clonus possible. In cases of functional neuroses these mechanical conditions must be provided in another way, i. e., by active muscular tension."

These observations coincide with mine, but I do not agree that this "active muscular tension" indicates a neurotic or hysterical element. As mentioned above, some of the cases of paralysis agitans and neurosyphilis showed electromyograms of this type. In the case figured there was only a slight tremor when the leg was relaxed, but when

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[9] Cobb: Johns Hopkins Hosp. Bull. **29**:250, 1918.

[10] Johns Hopkins Hosp. Bull. **29**: 1918, plate 35.

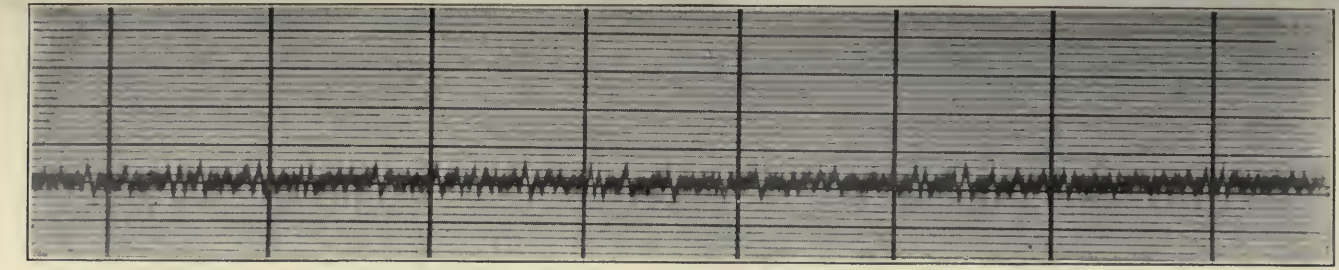

Fig. 1.—Tonic hysterical contraction: Electromyogram from left rectus femoris in Case 1, showing steady tetanus-like stream of action-currents at a frequency of 64 main waves per second. The perpendicular lines divide the illustrations into periods of ⅕ second.

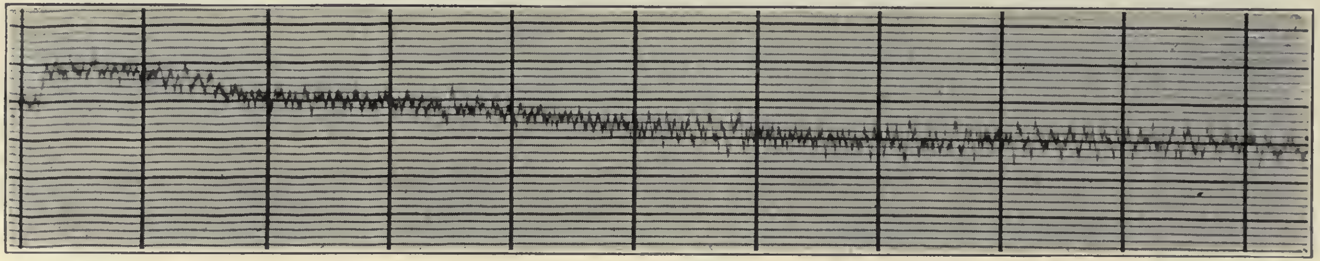

Fig. 2.—Tonic hysterical contraction: Electromyogram from left tibialis anticus in Case 2, showing a steady series of action-currents at a frequency of 60 main waves per second.

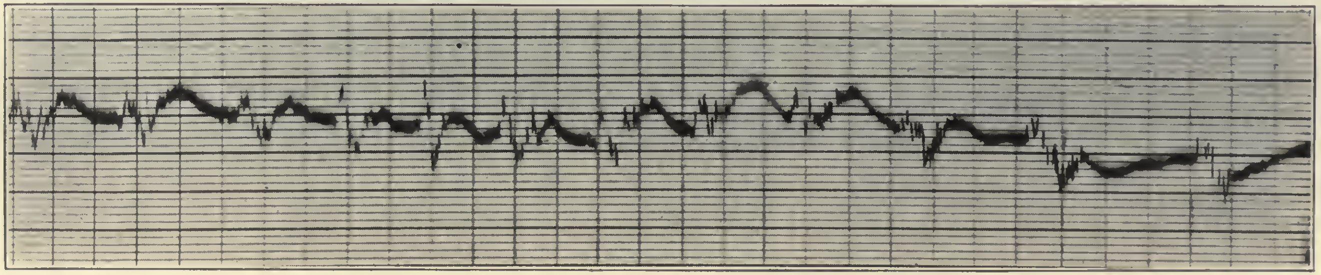

Fig. 3.—Clonic hysterical contraction: Electromyogram from Case 3. The short groups of action-currents occur periodically. The frequency of these action-currents is about 45 per second. The periods between contractions vary from 0.4 to 0.8 of a second, thus the rate averages 1.6 per second. (The plate was moving too slowly to give good detail and the focus was not sharp toward the end, necessitating retouching.)

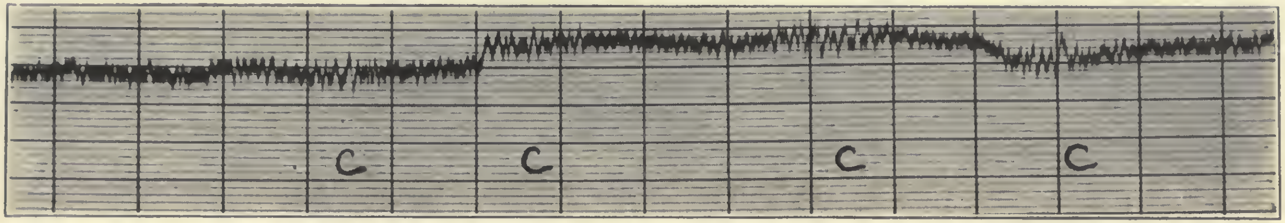

Fig. 4.—Periodic hysterical contraction: Electromyogram from Case 4. The steady stream of action-currents is due to the voluntary extension of the arm, but at points *c, c.* there is seen an increase in the size of the action-currents due to the involuntary movement, which appears at periods of from 0.58 to 0.72 of a second, i. e., at an irregular rate of 1.5 per second.

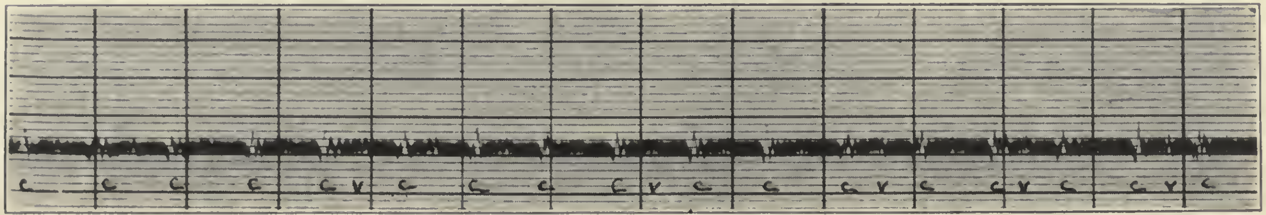

Fig. 5.—Paralysis agitans: Electromyogram from gastrocnemius. There is a regular periodicity giving a tremor rate of 6.2 per second, each tremor contraction being accompanied by a group of action-currents (*c, c, c*). Between these is a slight electrical activity (*v, v, v*) probably due to the voluntary tension put on the muscle to elicit the tremor.

the weight of the leg was balanced on the ball of the foot the tremor was brought out conspicuously (Fig. 5). In other words, a slight voluntary tension of the muscles often serves to bring out a tremor or clonus that otherwise could not be seen. Many normal people develop a clonus after taking coffee, or when fatigued,[11] by putting their muscles on just the right tension. This slight voluntary contraction underlying the grosser involuntary tremor seems, then, to depend rather on the excitability and threshold of the comparatively simple reflex arcs than on the more highly integrated psychologic levels which are concerned in the pathology of hysteria; for example, a person apparently normal and with no symptoms of hysteria may have hyperactive reflexes and clonus. Between this clonus, that needs slight voluntary muscular tension for its elicitation, and the clonus of a spastic paraplegic, there is no essential difference, for all intermediate graduations may be found. Consequently I should classify Salomonson's case not as an hysterical clonus, but as a clonus elicited in a person with hyperactive reflexes when she put her muscles on a slight voluntary tension.

In interpreting the electromyogram of a tremor, therefore, hysteria should be suspected whenever the contractions registered show conspicuous irregularity of rhythm, and when they occur with a periodicity not commonly found in the typical tremors of known clinical syndromes.

CONCLUSIONS

1. The electrophysiology of an hysterical muscular contraction is similar to that of a normal voluntary muscular contraction.
2. The short, repeated, hysterical contractions that clinically resemble tremors can be differentiated electromyographically from such a tremor as that of paralysis agitans, or from clonus, by their slower rate and greater irregularity.

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[11] Levi: Arb. a. d. Neurol. Instit., Wien Univ. **2**:28, 1917.

DOUBLING OF THE SPINAL CORD*

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Variations in the gross anatomy of the spinal cord are unusual. Observation of many spinal cords, in our regular class dissections during a period of five years, tends to confirm this statement. During this period one double spinal cord was found. A careful search of the literature failed to disclose the report of a case similar to the one I describe. Although it was impossible to obtain a clinical history, the variation is so unusual as to be of more than casual interest to the clinician, the surgeon and the anatomist.

This rare variation was encountered during the regular laboratory work in the dissecting rooms at the Daniel Baugh Institute of Anatomy of the Jefferson Medical College. Unfortunately the spinal cord was not in a state of preservation to admit microscopic study of serial sections.

The specimen was obtained from an adult white male cadaver, well nourished, about 35 years of age and of unusually good physique. A careful external examination showed no abnormalities. The vertebrae were normal and there were no abnormalities of the bony canal, either ventral or dorsal to the spinal cord. The skin and overlying tissues of the lumbar and cervical region were likewise without abnormalities.

On opening the dural sac, an unusual variation of the spinal cord and inner surface of the dura was discovered. The spinal cord was divided in its caudal portion into two distinct symmetrical halves, the two entirely enclosed in a single dural sheath as shown in the illustration. Similarly, the arachnoid stretched across as a single membrane. The bifurcation began at the caudal border of the eleventh thoracic vertebra and extended to the caudal limit of the cord, forming two independent symmetrical halves. The caudal limit of each half was at the level of the cephalic border of the first sacral vertebra. The conus medullaris of each half was independent and at the level of the third sacral vertebra, they united into a single large filum terminale, the latter coursing caudally for a distance of 5 cm. to gain attachment to the dorsal surface of the first segment of the coccyx.

The cervical and thoracic portions of the cord were normal in appearance. The peripheral nerves cephalic and caudal to the bifurcation were carefully dissected and presented no variations in the formation of the different plexuses and in their normal peripheral distribution.

The inner surface of the dura at the level of the caudal border of the twelfth thoracic vertebra likewise showed an interesting and rare anomaly. Where the division of the cord was total, a distinct band of dura was found projecting between the two halves of the cord. The band of dura was directed ventrodorsal and had a first attachment of 1 cm. on both the ventral and dorsal ental surfaces of the dura. It was of an hour-glass shape and was placed in the sagittal plane. This band or fold was clearly a part of the dura, being of the same texture and consistency. In the unopened dura it seemingly caused no pressure on the halves of the cord. The division of the spinal cord began 0.5 cm. cephalic to dural band noted above.

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There were also present, as shown in the illustration, a number of small flattened calcareous plaques adherent to the spinal arachnoid; the largest was about 7 mm. in its long diameter. Each plaque appeared to be accompanied by a small artery. The calcareous plates will be dealt with more in detail in the discussion of this paper.

The unusual features of this specimen are: first, the presence of a bifurcated spinal cord beginning at the twelfth thoracic vertebra; second, the presence of an hour-glass-shaped dural band directed in the sagittal plane and firmly attached to the ventral and dorsal ental surfaces of the dura, which entered the dorsal median fissure and completely divided the cord; third, the absence of other abnormalities that not infrequently accompany bifurcation of the spinal cord; fourth, the extreme caudal extent of the spinal cord—first sacral vertebra.

EMBRYOLOGY

Kunitomo,[1] in a careful study of a large number of human embryos, shows that in very young specimens the spinal cord reaches the extreme tip of the tail, and throughout its length is quite uniform in structure. He further calls attention to the fact that in the formation of the filum terminale in addition to the dedifferentiation of the caudal end of the medullary tube, there is also mechanical disproportion between the growth of the medullary tube and that of the vertebral column. Streeter's studies show that the filum terminale represents that portion of the spinal cord caudal to the second coccygeal segment. This strand elongates by interstitial growth in adaptation to the ascendinng displacement of the cord. The part of the cord that undergoes dedifferention into the filum terminale is caudal to the ventriculus terminalis (the fusiform dilatation of the central canal in the lower end of the conus medullaris). The caudal tip of the dural sac maintains its initial relation to the vertebra rather than to the definitive spinal cord, and remains attached to the filum terminale in the sacral region at a more or less fixed point. Early in the embryo the spinal cord and the vertebral column lie alongside of each other in a metameric manner, corresponding in position segment for segment. Owing to the disproportion in growth between the spinal cord and the vertebral column, relative displacement of their respective levels occurs. In the first twenty-five weeks there is an ascent of the ventriculus terminalis from the level of the second coccygeal vertebra to the third lumbar, and

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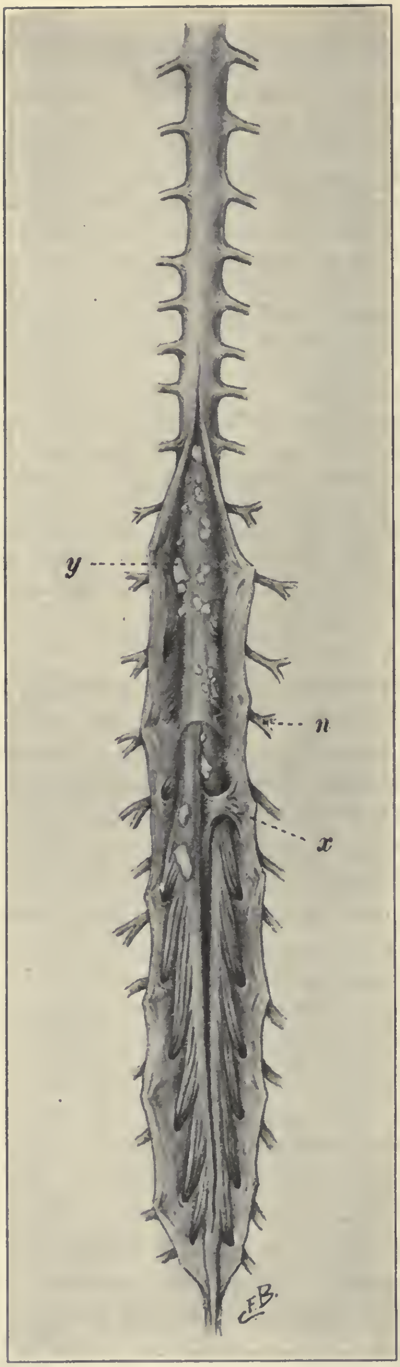
1. Kunitomo, K.: Contributions to Embryology, Vol. 8, Publication No. 271, Carnegie Institute of Washington **8:** 1918.



The lower part of the dural sac has been opened and turned aside. The arachnoid with the superimposed calcareous plaques is shown stretching across the spinal cord to the level of the eleventh thoracic spinal nerve indicated at N. The arachnoid below this level has been removed to show the dural plaque projecting between the halves of the spinal cord. Y indicates a calcareous plaque; N the eleventh thoracic nerve and X the dural plaque.

there remain but two segments before the adult position is reached. One may say that the principal part of the migration of the cord is accomplished during the first half of fetal life. It is thus evident that the adult position of the cord is the result of two factors — the dedifferentiation of the caudal portion of the neural tube into the filum terminale, and the disproportionate growth between the spinal cord and the vertebral column. Another factor may also enter into the formation of the filum terminale: The relation of the pia overlying the spinal cord is early established, likewise the caudal tip of the dural sac holds the same position in the embryo and the adult. It would seem plausible that as the cord ascends, due to the caudal growth of the vertebral column, the pia is gradually attenuated, interfering with the proper nutrition of the part of the spinal cord destined to become the filum terminale. The more caudal extent of the cord is, therefore, not due to a partial failure of the cord to ascend as is commonly thought, but is the result of a failure of a part of the embryonic undifferentiated cord to continue its retrogressive changes into the filum terminale.

Obviously, the length of the filum terminale depends on the caudal limit of the spinal cord. In cases in which the spinal cord extends more caudally than its usual limit, there is a corresponding decrease in the length of the filum terminale.

The neurenteric canal is a transitory communication between the neural tube and the primitive digestive tube. It is purely a temporary structure having normally completely disappeared by the end of the third week. Certain anococcygeal tumors are believed to arise from the neurenteric canal, as well as from the hind-gut. Such a canal is represented by a remnant passing from the rectum to the sacral region of the spinal canal. Keen, in 1906, reported a sacrococcygeal tumor associated with a spinal bifida and a sinus beginning at the skin, perforating the sacrum and the spinal canal, and terminating in the lumen of the gut.

The dural band present in this specimen seems to have been the factor that produced the division of the cord. I have been unable to explain the genesis of this variation on the basis of our known embryology of the cord and dura. The dural band appears to be due to a disturbance of the mesothelium resulting in an excessive mesothelial activity at this point with the projection of a fold of dura between the two halves of the cord.

LITERATURE AND DISCUSSION

Doubling of the spinal cord is a rare anomaly, and is the bifurcation of the medulla spinalis into two symmetrical or asymmetrical halves for a variable part of its course. The division usually occurs in the lumbar or lower thoracic segments of the cord. Hanover describes a case of complete division of the spinal cord with a distinct central canal in each half extending to the fourth ventricle. Frequently this variation is only one of many anomalies in the same subject. Doubling of the spinal cord has been found associated with rachischisis, myelomeningocele, hydromeningocele and anencephalus. Frequently the anomaly is associated with cutaneous and bony malformations.

A complete doubling of the cord is one in which two well-marked symmetrical halves are present; at times one half is rudimentary and the other well developed, or the two halves are asymmetrical. The spinal cord substance collectively is probably decreased when the division of the cord is well marked. In a division that is not well-marked, or in an otherwise nondefective cord, the gray and white matter of both halves are equal to the normal amount of spinal intramedullary substance. The central canal may be absent or may be present as a distinct canal in each half, varying in outline. Thirty-five cases of double spinal cord were collected by Steiner;[2] twelve were eliminated by Bruce, McDonald and Pirie[3] as being merely split cords and thirteen as artefacts, leaving ten cases of true doubling of the cord. Of the latter, seven were associated with spina bifida. In seven of Steiner's thirty-five cases the bony vertebral canal was normal. The age of Steiner's collected cases ranges from a 3 months' old fetus to an adult of 76 years. In Steiner's cases, eight occur in adults whose ages range from 19 to 76 years. In one case the doubling was limited to the cervical and upper thoracic segments; in another (Hanover's case mentioned above) the division involved the entire cord. All other cases occurred in the lumbar region. The case described by Steiner occurred in a female infant 3½ months old. A sacral swelling was present which fluctuated and when opened revealed an oval communication with the central canal. There was likewise a medial defect of the bony vertebral column consisting of a split 2 cm. long beginning on the first sacral segment and continuing through the sacrum and coccyx. The division of the cord began at the caudal border of the fifth lumbar vertebra, the cord continuing to the level of the third sacral vertebra as two independent halves. Each half resembled a normal cord.

There are cases reported showing diffusion and spreading of the gray commissure, but without division of the white matter. These are not true and complete divisions of the cord.

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[2] Steiner, F.: Ueber Verdoppelung des Ruckensmarks, Konigsburg, 1896.

[3] Bruce, A.; McDonald, S., and Pirie, J. H. H.: A Case of Localized Doubling of the Spinal Cord, Rev. Neurol. & Psychiat. **111**:709, 1905. A Second Case of Partial Doubling of the Spinal Cord, ibid., 1906, IV, 6-19.

Bruce, McDonald and Pirie report a case of doubling of the cord occurring in a woman, aged 31. There were no sensory or motor symptoms. In the lumbosacral region the cord divided into two distinct trunks entirely enclosed in a single dural sheath. The division was total and a fold of dura projected for a certain distance between the two halves not reaching the ventral surface of the dural sac. The cord was completely divided for about 2 inches. There were no abnormalities of the bony vertebral canal. In the case described in this paper the fold of dura projected between the halves of the cord and had a distinct attachment to the ventral and dorsal ental surfaces of the spinal dura.

Bernstein[4] has described a case of bifurcation of the spinal cord beginning at the first lumbar segment. Examination of sections of the cord showed two seemingly complete cords united slightly along their mesial aspects. Each half of the cord presented two anterior and two posterior cornua. Caudal to the third lumbar vertebra the two halves of the cord seemed to fuse again. The filum terminale was single and large. At the level of the second lumbar segment each half exhibited a distinct central canal.

Van Gieson has called attention to the fact that artefacts are easily produced simulating a bifurcation of the spinal cord and certain cases demand the most careful study to eliminate the doubt of an artefact. This statement is confirmed by Spiller in a study of a specimen of what seemed a double spinal cord. On more careful examination and study he decided it was an artefact.

Westphal[5] has reported an unusually interesting case of a lumbar division of the spinal cord occurring in a woman, aged 29. There were no associated abnormalities. On opening the dural sac the spinal cord was found divided in its caudal portion into two distinct halves, the left half being broader and larger than the right. The dura exhibited no abnormal variations. The left half presented in its distal portion a distinct cyst which was filled with glassy mucus. The walls of the cyst were whitish in color and spotted with yellowish red areas. The latter on close examination proved to be small vessels and small hemorrhagic spots. The cervical and thoracic segments of the spinal cord were normal in appearance. Deep in the dorsal median fissure of the spinal cord numerous smooth muscle fibers were demonstrable on microscopic examination. The strands of smooth muscle increased in number caudally, blending with a distinct swelling also composed of smooth muscle. The swelling presented in its caudal portion a lumen, cylindrical in outline and forming the walls of the cyst noted above. The mass of muscle fibers and the spinal cord were covered by the overlying pia. The right side likewise presented a swelling smaller than that of the left and similarly composed of smooth muscle. Some of the nerve fibers streamed irregularly through the cyst wall. The cyst was not a dilatation of the central canal as a distinct ependymal canal was demonstrable in each half of the cord at levels with the cyst. The walls of the cyst as noted were formed of smooth muscle and microscopic examination of serial sections revealed no nervous tissue or ganglion cells.

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[4] Bernstein, J. M.: Doubling of the Spinal Cord, Trans. Path. Soc. Lond. **7**:318, 1905-1906.

[5] Westphal: Ueber eine bisher ausscheinend nicht beschriebene. Missbildung am Ruckenmarke, Arch. f. Psychiat. **46**:712, 1906.

A number of swellings, myofibrolipoma in character, have been reported by Von Recklinghausen, Fischer, Von Ribbert and Marsh usually associated with lumbar spina bifida. In the case of Westphal the cyst was composed of smooth muscle fibers extending into the dorsal median fissure. The growth of smooth muscle fibers along the cord was first noticed by A. Pick. This interesting observation noted by Pick showed the presence of myomatous growths about blood vessels in the white and gray matter. Bonnet believes they represent a remnant of the hind-gut of the embryo occurring as an outgrowth from a rest of the hind-gut before the back is completely formed. Marchand and Von Recklinghausen show the relation between the neurenteric canal and the spina bifida. Middledorph comments on certain large intestinal tumors in the region of the coccyx as the persistence of the postanal opening. Muscatello has described a case of a fetal anomaly in which the lumen of the gut tract communicated by a winding canal directly with the central canal of the cord, confirming the observation of Marchand and Von Recklinghausen relative to the abnormal communication of the intestinal tract and the caudal portion of the ependymal canal. The latter is to be particularly remembered in spina bifida. Tillman states that growths in the coccygeal region may occur as rests of the medullary tube, the chorda, intestine, muscle and skeleton.

Mention was made in a description of this specimen of the occurrence of a number of calcareous plaques adherent to the spinal arachnoid. Calcareous deposits within the central nervous system are most common on each side of the superior longitudinal sinus as the result of calcium deposits in the arachnoidal granulations or villi (pacchionion bodies). The writer has observed the occurrence of macroscopic calcareous arachnoidal plaques in approximately 10 per cent. of all cadavers. The deposits, acording to Cushing and Weed,[6] are composed of true bone as is evidenced by the occurrence in them of the typical lacunar system, Haversian canals and the characteristic osteoblasts and osteoclasts. Another example of bony deposition is the occurrence of an osseous nodule in the falx cerebri. The observation of the dura and its reflections in our neuro-anatomy laboratory frequently show this example of bony deposit, in reality osteophytes and practically always confined to the falx cerebri. Calcareous deposits appear to be a normal physiologic process in two structures of the central nervous system, the corpus pineale and the plexus choroideus. Virchow called attention to the occurrence of lime salts in the cerebrospinal axis in that type of cerebral neoplasm which he designated psammome. Weed and Cushing have noted the occurrence of a thickened arachnoid covered with plaques of true bone in a patient suffering from epilepsy. He had, in early life, been a victim of severe infection.

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[6] Cushing, H., and Weed, L. H.: Studies on the Cerebrospinal Fluid and Its Pathway, No. IX. Calcareous Deposits in the Arachnoidea, Johns Hopkins Hospital Bulletin **26**:367, 1915.

Weed, in a study of a series of brain tumors, found a deposition of calcium salts in 40 per cent. of all cases. They have been described in all slowly-growing cerebral neoplasms. Bony growths in the form of osteophytes arising from the meninges are of frequent occurrence and have been repeatedly recorded.

In conclusion, it is interesting to comment on the possible effect of a lumbar puncture in a patient with a cord extending caudal to third or fourth lumbar vertebra. The author is not willing to venture an opinion because of the rarity of its occurrence, yet it seems a fair assumption that injury to the cord would follow with its resulting symptoms. It is not far-fetched to state that occasional untoward effects, such as paralysis following lumbar puncture, may be due to the presence of a cord extending to levels below its normal caudal limit.[7]

My thanks are due to Dr. J. Parsons Schaeffer, head of the Department of Anatomy, for permission to report this specimen and for his interest and criticism during this study.

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[7] In addition to the references given, the following may be of interest:

Stewart, J. P., and Bernstein, J.: A Case of Partial Doubling of the Spinal Cord, Rev. Neurol. & Psychiat. **4**:729, 1906; Streeter, Geo. L.: Factors Involved in the Formation of the Filum Terminale, Am. J. Anat. **25**: No. 1.

AN ACUTE DESCENDING RADICULAR TYPE OF EPIDEMIC ENCEPHALITIS

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INTRODUCTION

During the recurrence of epidemic influenza this winter, there have appeared a number of cases which at first seemed to be a new type of infection of the central nervous system. These have assumed a characteristic course, and should perhaps be considered a new clinical syndrome; however, from an etiologic point of view, it must of necessity be regarded as a distinct form of epidemic encephalitis. Five of these patients have come under my observation, and their recovery necessarily limits this study to the results of a clinical investigation.

Radiculitis has been described clinically, especially in conjunction with herpes zoster, syphilis, and as a secondary manifestation resulting from pressure on the dorsal roots; but as an inflammatory process progressively involving the spinal roots it is rarely encountered, and has not been described.

According to Mayer,[1] radiculitis is usually a unilateral condition, and is rarely bilateral except in the later part of its course.

Déjerine,[2] in his "Syndrome radiculaire," describes a symptomatology essentially of the nature of those noted below. He mentions the frequency of localized radiculitis in the lumbosacral and brachial segments (C 5 to C 7), and its rarity in other sections of the cord, remarking "that rarest of all it may exist in a more or less generalized form." According to Déjerine, this syndrome has for its clinical manifestations pain, hyperesthesia and paresthesia, corresponding to the dermatomes. A herpetic eruption may also be present. The deep reflexes may at first be exaggerated and later diminished, and there may be muscular atrophy and spasm of the muscles.

The clinical picture which I shall describe shows generalized distribution and presents the above "syndrome radiculaire" of Déjerine.

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[1] Mayer, Edward E.: Radiculitis; Its Diagnosis and Interpretation, J. A. M. A. **71**:353 (Aug. 3) 1918.

[2] Dejerine, J.: Semeiologic des affections du systeme nerveux, Paris, 1914, **2**:820 and 884.

SYMPTOMATOLOGY AND PHYSICAL SIGNS

The clinical course of this condition has been stereotyped in its symptoms, which have manifested themselves in a typical descending progression beginning in the higher cervical and terminating in the sacral region. The chief symptoms are: (1) sharp lancinating root pains, (2) paresthesia, (3) muscular spasms, (4) hyperesthesia, (5) delirium, and (6) fever.

The symptoms develop thus: The patient begins to complain of slight paresthesia in the back of the neck or shoulder; this is represented by a feeling of "threads" or a "puckered" sensation in the skin. Within twenty-four hours sharp shooting pains develop in the back of the neck or shoulders, and then progress downward into one or both arms. These pains are severe, sudden or knife-like in quality, and intermittent, suggestive of the "lightning" root pains of tabes, though of greater intensity. They prevent sleep and are relieved only by morphia.

Paresthesia of all varieties accompanies the pain, including numbness, tingling, burning, formication and "pins and needles" sensation. They are constant and distressing. An intense hyperesthesia to all sensation may develop over some one of the involved areas, perhaps in one or both arms, or, as in the case of one patient, in a small area in the suboccipital zone. This hyperesthesia usually is quite general, involving as well the anterior and posterior division of the affected zone.

Several days following the onset of pains, involuntary muscular spasms appear, causing coarse twitching movements of the head, shoulder and neck, which remind one of the spasmodic contractions occasionally occurring in spinal cord tumors. These contractions are present during sleep. One of the patients showed spasm of the diaphragm, which at first was unilateral, and later became bilateral, resulting in grunting speech. When these spasms affect the arms it is almost impossible for the patient to feed himself.

After a week the symptoms become much less severe in the arms and a mild delirium begins to develop. The mind becomes somewhat disoriented; the patient has mild hallucinosis and delusions, and shows considerable mental confusion. This acute psychotic state lasts for three or four days, followed for about two weeks by dulness and lack of interest almost to the point of apathy. No real drowsiness or lethargy was noted at any time in any of the cases.

The disease is sometimes ushered in by a chill followed by a persistent, low fever varying from 99 to 101 degrees, occasionally rising as high as 102 to 103 F. There is no acceleration of the pulse.

The clinical course of the disease is peculiar. Following symptoms in the arm and neck, it progresses downward in an orderly manner. Pain radiating in type around the upper chest develops, then girdle sensations and the tightness around waist. Pain and sensory disturbances in the arms become less severe, and spasmodic twitching movements of one or both abdominal recti or transversalis muscles appear. From the onset of symptoms until the severe arm pains cease about eight or ten days elapse. Then the symptoms confine themselves largely to the intercostal and abdominal regions for two or three days. There is generally delirium at this stage, but the pains are usually less severe. The symptoms then descend to the lower extremities; there is a tremendous recrudescence of the pain which extends down the legs with shock-like intensity, requiring an opiate for relief. Paresthesia and muscular spasms involve the legs. One patient suffered so intensely from the hyperesthesia over the sacral "saddle area" that she could hardly sit in bed. A slight increase of the fever precedes the invasion of the lumbosacral region, and after four or five days there is a return to a 99 to 100 degree temperature. The course then becomes more subacute accompanied by mild fever, a moderate number of light, fleeting root pains and some paresthesia. The patient progresses toward a slow convalescence of many weeks' duration. At this time the twitchings in the arms and legs have resolved themselves into an intention type of tremor which is coarse and oscillatory, and disappears when the patient is at rest.

At no time during the course of the disease are there any vasomotor, trophic or sensory changes other than those mentioned above. There is no anesthesia to touch, pain or temperature, no disturbance of deep sensibility, and no herpes. The reflexes are at no time profoundly altered. In the patients observed, a slight exaggeration of the deep reflexes was noted at the onset of the disease with a diminution in their activity several days after the invasion in each region. Toward the convalescent period in each case there developed some weakness of the grasp of hand and a beginning atrophy in the musculature of hands and arms, which seemed more rapid than could be accounted for by disuse. None of the patients showed alteration in pupillary reaction or any blurring of vision or oculomotor weakness. The control of the bladder and rectum was at all times retained.

One symptom of interest, noted in all patients, was an involuntary flexion of the head. The flexion was not a necessity for the patient's comfort, for motion did not increase or produce pain. This position of the head reminds one of the attitude seen in a cervical spinal cord tumor, syringomyelia, and sometimes in amyotrophic lateral sclerosis.

COURSE AND PROGNOSIS

The predominant symptoms of the syndrome justify the belief that the lesion is due to an acute descending inflammation progressively involving the dorsal roots. The onset is sudden, the pain beginning in the upper cervical region, remaining there for from nine to eleven days, then gradually descending to the chest and abdomen, remaining there for about three days. During this time the patient develops an acute psychosis. The symptoms clear above as they progress downward. The legs are next involved, and finally the sacral region. At the end of about two and a half weeks the symptoms reach a more or less stationary stage with low fever and mild, fleeting general radicular pain and paresthesia. These persist for a longer or shorter period, and the acute symptoms eventually disappear.

The convalescent stage was most characteristic in Case 4. The patients have a few pains and some paresthesia; the arms are weak, with slight muscular atrophy of the hands and arms. They are considerably fatigued and appear somewhat depressed over their long illness.

The ultimate prognosis cannot at this time be estimated, as the patients have not been observed for a sufficient period to determine the possibility of the later appearance of signs of spinal cord destruction resulting from the acute process.

LABORATORY FINDINGS

Examination of the blood revealed a moderate leukocytosis, between 12,000 and 14,000, and only a slight polynucleosis, 72 to 84 per cent. Urinary reports showed no changes, and blood cultures were negative. Spinal fluid examination in three cases revealed no increase in pressure; it was clear and colorless. The cell count was increased, 31 and 40 cells in two cases, while the third was within normal limits. All of the cells were lymphocytes, no polymorphonuclears being seen. Globulin was increased in two cases; one case showed a negative Noguchi reaction. Wassermann tests on both blood and spinal fluid were negative, and the colloidal gold curve was of the "irritation type" in two instances.

REPORT OF CASE

Case 1.—J. McG., aged 39, elevator man, seen on Jan. 22, 1920, at St. Luke's Hospital.

Family and Past History.—This included no diseases bearing on the present condition.

Present Illness.—Following exposure ten days before (January 12) the patient first noticed intermittent sharp pain shooting down the right arm, which soon passed into the left arm. These pains, which became more frequent and intense, were accompanied by numbness, tingling in the arms and hands, and a feeling as though the right arm were continually "asleep." Spasmodic twitching movements of the muscles of the arm and upper chest appeared soon after onset, and a spasm of the right half of the diaphragm, causing grunting respirations and a catch in the speech. The afternoon I saw him the condition was very much as described above, except that both halves of the diaphragm were twitching and the pains in the arm were beginning to abate. The temperature was 99 degrees, and an acute delirium had begun with visual hallucinations and attempts to get out of bed to go to a funeral.

Physical Examination.—The patient was a well nourished man, acutely ill, who appeared "toxic" and was delirious. The pupils reacted actively to light, were regular and equal. There was no facial asymmetry, no oculomotor palsies, and the tongue did not deviate. There were no disturbances of gait, no weakness of arms or legs, and no tenderness over nerve trunks. Reflexes: Triceps, biceps and wrist jerks were somewhat sluggish, and the abdominals were not obtained, perhaps on account of distention. Knee and ankle jerks were present, and there were no clonus or plantar extensor responses.

Sensorium: Over the posterior aspect of the neck above the seventh cervical spinous process there was a definite area of hyperesthesia extending upward to the postoccipital region; no other disturbances were noted. The temperature had varied up to this time between 99 and 100 degrees. The urine showed acetone and diacetic acid, which soon disappeared.

Spinal puncture the following day revealed 31 cells, all lymphocytes; globulin +; and the Wassermann reactions were negative here as well as in the blood.

Course of Disease.—This was most prolonged.

January 23: The patient was delirious with severe pain in the head, back of the neck and arms. He said that his pillows felt like "blocks of wood." Spasms of the abdominal muscles were first noted.

January 24: Pains were were first noticed in the legs and feet, especially in the right leg. There was mental confusion and the spasmodic twitchings were still present.

January 25 and 26: The patient complained of severe pains shooting down the legs. There was some twitching of legs and he tore the bed clothes. He obtained no relief from codein. Mentally, he appeared to be more clear. The reflexes in the arm were still sluggish. Abdominal reflexes were present. The right knee jerk was diminished; the left more active. There was no Babinski reflex or ankle clonus.

January 31: The patient was somewhat confused at times, but felt much better. No spasmodic twitchings were noted and he had infrequent mild shooting pains in the legs. No morphin has been necessary since January 29. The head was stooped. Paresthesia persisted in both arms and legs.

February 7: During the past four days he had had some slight neuralgic pains in both arms and legs, weakness of grips and slight atrophy of the forearm and hand muscles were present. Mental confusion had disappeared. Occasionally he had numbness in the extremities.

February 17: The patient sat up in a chair. He complained of pain in the right leg and back of the neck. He had profuse diaphoresis.

March 8: During the past few weeks there had been gradual progression toward recovery. He had had some pain from time to time, but it had not been distressing.

The temperature during the first week after admission was from 100 to 98 degrees; second week, from 98 to 100 degrees; third week, from 99.2 to 100.2 degrees; fourth week, 99 to 99.4 degrees; fifth week on, 98 to 99 degrees.

The urine and blood Wassermann test were negative.

The blood counts were: January 14, red blood cells, 4,400,000; hemoglobin, 93 per cent.; white blood cells, 7,200; polymorphonuclears, 70 per cent.; lymphocytes, 30 per cent. February 1: White blood cells, 14,500; polymorphonuclears, 78 per cent.; lymphocytes, 22 per cent.

The spinal fluid was examined but once, and the colloidal gold curve showed 111.51.51.510.5000.

This patient showed throughout the course of the disease more clouding of consciousness than any of the others. There was at one time almost a lethargy, but no somnolence was noted. The spasmodic twitching of his diaphragm was conspicuous; otherwise his progress was quite characteristic.

Case 2.—F. B., aged 21, a woman, with no occupation, was seen on Jan. 29, 1920, four days after admission to St. Luke's Hospital.

Family and Past History.—This was negative, except for a chorea at the age of 6, and the presence of a hypertension which has been known for some years.

Present Illness.—The onset on January 19, was sudden, following exposure during an automobile ride the same morning. The illness began with sudden, severe, shocking and knife-like pains in the right shoulder, soon spreading down to the hands and to both shoulders. This was accompanied by paresthesia, numbness, tingling and extreme hyperesthesia to light touch. No herpes was present and the patient thought there was slight fever. Two days later spasmodic twitching movements of the muscles of the arms developed. They seemed to be initiated by the pain, and were so distressing that they prevented sleep and necessitated frequent doses of morphia. There had been an excessive amount of gas in the stomach, but no diaphragmatic spasms. In the last few days before being admitted to the hospital the hyperesthesia had left the right arm and spread upward so as to include the left posterior occipital region in which the pain was now severe, and there was a spasm of neck muscles drawing the head to the side. Paresthesia persisted in the hands and arms.

Physical Examination.—The patient was a well developed girl, apparently suffering considerable pain and complaining of lack of sleep. The head was held somewhat bowed. There was perhaps a slight lack of facial expression. There were no oculomotor or pupillary disturbances; no asymmetry of face or deviation of tongue. The lungs and heart were normal. The blood pressure was: systolic, 250; diastolic, 200. The abdomen was negative, except for slight distention. Muscular strength was good, except for some weakness of grip of both hands. Reflexes: Biceps, triceps and wrist were moderately diminished. Abdominals were present. Knee jerks and ankle jerks were exaggerated and plantar flexion was present. There were no clonus or extensor responses and no ataxia, but there was a coarse tremor of the arms and myoclonic movements of arms and of the head on the neck (trapezius and sternomastoid).

Sensorium: There was no anesthesia, and over the entire left arm extending to the midline anteriorly and posteriorly and up over occiput on left side so as to include cervical 3, 4, 5, 6, 7, 8 and T 1, 2 and 3 dermatomes, there was exquisite hyperesthesia causing the patient to wince at the touch of the finger or pin. There was no herpes.

Course of Disease.—January 27: The back of the neck, head and shoulders were painful, and paresthesia of the hands persisted.

January 30: A rise of temperature occurred from 100 to 102.3 on January 31, reaching a level of 100 degrees on February 1.

January 31: There was almost no pain in the arms, and the hyperesthesia had disappeared. She had been more comfortable for the past forty-eight hours, but there was still slight impairment of grips in the arms and some twitching. Flexion of the head was noticed. Paresthesia in the ankle, which was complained of on January 30, was increased, and there were also a few mild shooting pains down legs.

February 1: There were severe knife-like root pains in the legs with paresthesias and a hyperesthesia of the toes and balls of the feet. Twitchings of legs had also developed. She was delirious and out of bed at times. Morphin was required constantly for relief of pain.

February 2: Pain had located in the thighs and buttocks.

February 7: Great improvement had occurred during the past three days, though there were still occasional mild pains in both extremities and some in the knees.

February 10: Pains in the legs were moderate, not requiring morphia. There was intense hyperesthesia over the buttocks corresponding to sacral second, third, fourth and fifth dermatomes. The coarse tremor of the arms was still present, and strength was returning to the grip. The patient left the hospital against advice, but to date is slowly recovering.

The temperature from the first week after admission was between 100 and 102.3 degrees, the latter figure representing the apex of the rise ushering in the involvement of the legs. The temperature during the second week was between 99.2 and 101 degrees; third week, from 99.1 to 100.3.

Laboratory Findings.—The blood pressure showed the following readings: systolic, 250; diastolic, 200; systolic, 200; diastolic, 150; systolic, 172; diastolic, 130.

January 25 the urine showed a specific gravity of 1.020; acid reaction; marked trace of albumin; sugar negative; few hyaline casts and red and white blood corpuscles in moderate numbers. These findings connote a nephritis of some severity.

Blood culture was negative.

Blood Wassermann test was negative.

The blood counts were, January 25: red blood cells, 5,000,000; hemoglobin, 103 per cent.; January 28: white blood cells, 12,300; polymorphonuclears, 78 per cent.; lymphocytes, 22 per cent. White blood cells, 11,200; polymorphonuclears, 80 per cent.; lymphocytes, 20 per cent. January 30: White blood cells, 6,500; polymorphonuclears, 82 per cent.; lymphocytes, 18 per cent. February 5: White blood cells, 16,500; polymorphonuclears, 84 per cent.; lymphocytes, 16 per cent.

The spinal fluid, January 25, showed 4 cells, all lymphocytes; the globulin was negative; the Wassermann reaction was negative. Colloidal gold curve: 1110.5000000.

The spinal fluid on February 4 showed 6 cells, 100 per cent. lymphocytes. Globulin and Wassermann tests were negative. Colloidal gold curve: 1111.5 10.50000.

This patient presented the characteristic picture of this syndrome. The few signs of thoracic involvement (chest and abdomen), and a delirium of short duration were noteworthy. Neither examination of the spinal fluid showed pleocytosis, although the colloidal gold test showed a definite irritation curve. She showed no cerebral signs, except the delirium and a certain lack of facial expression.

Case 3.—J. B., a colored woman, aged 32, married, was admitted to the City Hospital, Feb. 2, 1920, on the medical service. She was transferred to the neurological service Feb. 3, 1920.

Chief Symptoms.—These were twitchings of the body and pain in the shoulders, chest and abdomen.

Present Illness.—This began two weeks before the patient was admitted to the hospital after she had caught cold while working in a laundry. Pain began in the back of head and neck and three days later involved the arms with severe shooting pains, requiring an opiate for relief. Accompanying the pains were definite paresthesias, such as numbness and "pins and needles" over the shoulders, arms and hands, and hyperesthesia so intense that she could not stand the touch of her clothes against her. Her arms began to twitch at this time. She was taken to a hospital and in four days improved so much that she was sent home. The pain gradually left the arm and shifted down to the chest, and two days before admission to the City Hospital involved the abdomen with radiating pain to the front and with spasms of the abdominal muscles. The pain in the legs became so severe that she again went to the hospital.

On admission she was screaming with severe intermittent lancinating pain in the legs and complaining that her legs were numb and asleep. She was delirious, crying for relief, trying to get out of bed and the window, cursing and complaining that people were hanging over the bed when no one was near.

Physical Examination.—The patient was a well nourished colored woman, apparently delirious, and suffering from intermittent pain. There was no ptosis or diplopia; the pupils reacted actively to light and were regular and equal. There was no facial asymmetry or deviation of tongue. Heart and lungs were negative. There were spasmodic twitching movements of both recti muscles occurring at intervals and a coarse almost spasmodic tremor of the hands. There were weakness of muscles and no tenderness over the nerve trunks. Reflexes were present in the arms and legs and not altered. There was no Babinski reflex, no clonus or ataxia of arms and legs.

Sensorium: Over the back from the spine of the scapulae down to the sacrum there was hyperesthesia to light, touch, and pin prick that came well forward to the abdominal walls. The temperature varied between 99 and 100 degrees, and the pulse coincided.

Owing to the noisy delirium the patient was transferred to the psychopathic ward and so passed from detailed observation, also preventing any laboratory examinations from being made. Three days later I saw her again, and at that time she had come out of the delirium, was quite oriented, but was still suffering with pain in the legs. The spasms had disappeared, and she complained that the pressure of the bed clothes on her legs and feet was painful, and of a burning feeling in the legs.

Examination at that time showed a disappearance of the hyperesthesia over the back, no twitchings in the recti muscles, a coarse tremor of the arms and slight spasmodic twitchings in the legs at times. Over both legs from the knees to the toes on the outer aspect corresponding to dermatomes L 4 and L 5 there was intense hyperesthesia to all sensation. The temperature was 100 degrees.

No spinal puncture had been made and she felt distinctly improved. She left the hospital two days later against advice and passed from further observation.

This patient presented the syndrome of a typical descending radiculitis accompanied by mild fever and a delirium. Although her further progress was not known and no laboratory data were available, the course of the disease while she remained in the hospital was characteristic in all respects.

Case 4.—L. B., aged 56, an Italian baker, was admitted to the Third Division of the Neurological Institute Feb. 13, 1920.

History.—The patient had always been well until four weeks before. On January 19, he suddenly felt a shock-like pain in the little finger of the left hand which traveled up the arm and then included the right arm, easing a little in the left. Arms and hands felt numb, heavy and "swollen," and there was an intense hyperesthesia of the arms, becoming so severe at the back of the neck that he could not endure the touch of the pillow against the skin. Spasmodic twitching developed in the arms, and accompanied the shooting pains. The patient suffered so much that morphin had to be administered continually. He was restless and could not sleep. After about ten days to two weeks the symptoms progressed to the chest and then a constriction about the waist was complained of with radiating pains and spasm of the recti muscles. At no time was fever noted by the family, and there was no delirium, but restlessness was a prominent feature during the first ten days. After the pain left the arms, he did not suffer; even when the paresthesia and pain progressed to the legs, he did not complain a great deal. For ten days before entering the hospital he had been sleepy and drowsy, but at no time were blurred vision, diplopia, ptosis or herpes present.

On admission he complained of pain and stiffness in the back of the neck, and his head felt heavy. There was a tremor of the hands, some weakness of both hands and arms, and scattered fleeting pain throughout legs and body.

Physical Examination.—The patient lay quietly in bed and did not look ill. There was a perceptible lack of expression about his face when in repose. External ocular movements were normal; the pupils were equal, regular and reacted to light. There was no tremor of the face. Heart, lungs and abdomen were negative. The teeth show marked pyorrhea. There was no speech disturbance. The strength of the arms was slightly impaired and the grip was a little weak. There was a coarse oscillatory tremor of the hands and arms which at times seemed to involve the legs as well. This was somewhat of an "intention" type. There appeared to be slight atrophy of the hands and forearm muscles. The reflexes in the arms were somewhat sluggish. Knee jerks and ankle jerks were exaggerated. Plantar flexion was present. No sensory disturbances were present.

The patient did not have fever.

Laboratory Examinations.—Blood and spinal fluid Wassermann tests were negative. The blood examination revealed: red blood cells, 4,600,000; hemoglobin, 83 per cent.; white blood cells, 13,000; polymorphonuclears, 73 per cent.; lymphocytes, 27 per cent.

March 9: White blood cells, 12,000; polymorphonuclears, 56 per cent.; lymphocytes, 44 per cent.

The spinal fluid showed 40 cells, all of which were lymphocytes. The globulin was positive; colloidal gold curve: 1122200000. The withdrawal of fluid caused an improvement in the patient's condition. The second examination of the sppinal fluid on March 6 showed 37 cells, 100 per cent. lymphocytes; globulin +.

Althought this patient was seen after all the acute symptoms had subsided and with no facts except those which the wife was able to give, we have, nevertheless, a typical picture of descending radiculitis, the characteristic tremor remaining and a pleocytosis in the spinal fluid. The patient was making slow progress toward a complete recovery.

CASE 5.—E., aged 15, was a schoolboy. For the privilege of reporting this case I am indebted to Dr. W. H. Glafke, who saw it in consultation, and kindly furnished me with the following data:

History.—On January 9, without any previous disease, such as influenza, he came home from work complaining of a little headache. On January 10, while in New York with his mother, he complained of slight pain in his shoulder, which he thought was worse when he rode on the subway. When he arrived home he could not take his coat off easily, and complained that he could not use his right arm. It was not really paralyzed, however, as he swung the arm back and forth "in order to stir up the blood." The next day he had severe pain through the right side of the head, back of the ear, and on the next day his right upper jaw ached and the teeth felt cold. During the following day or two the pains traveled downward into the neck, shoulder and trunk, with extreme pain running around both sides of the body. The pain was spasmodic in character and was associated with visible jerking and contraction in the trunk muscles. He was extremely restless. After four days of this the leg muscles became involved and he was constantly stretching his legs, complaining of drawing sensations and pain in them. He had had no temperature up to this time, but now he developed slight irregular fever up to 100 degrees. He became restless and noisy after being removed to the hospital, begging to be taken home. Blood tests taken then were reported by the family to be negative. After four or five days in the hospital he was removed to his home and became delirious about the twenty-first. He would speak and answer questions correctly, but then would lapse into a state of muttering delirium, complaining that the Bolsheviki were after him, and his eyes would rove about in a frightened way, apparently as the result of visual hallucinations. He could not sleep at all in spite of chloral and bromids. I (W. H. G.) saw him on the twenty-third. His temperature was running up and down from 102 degrees. He was still delirious. His pupils reacted to light and were equal. His neck was not stiff; in fact, the neck muscles were inclined to be paretic and his head dropped forward most of the time. He was not complaining of pain. The lungs, heart and abdomen were negative. He was generally very restless, and there was considerable gross tremor and twitching of all the muscles of the arm, trunk and legs. He had a generalized rash over the body (probably from bromids, and not of herpetic type). A spinal puncture was not done at any time. On January 29 his mother wrote that he was somewhat better, and was less delirious. The jerking of the muscles had ceased, and he was complaining of pins and needles all through his body. The temperature was 101 and the pulse 120. He was beginning to get a few hours' natural sleep at a time. The attending physician felt that he was slowly recovering.

This case presents the typical syndrome of the disease with descending progression of the symptoms from above downward with accompanying delirium. An exceptional feature in this patient was the pain and paresthesia in the upper jaw and teeth, evincing an involvement of the second branch of the gasserian ganglion.

DISCUSSION

The syndrome described accompanies an acute infectious disease producing symptoms largely spinal in localization, except for the delirium. We are dealing, therefore, with a lesion chiefly affecting the meninges of the cord and especially involving the posterior roots in a progressive descending inflammation.

The appearance of these unusual cases was almost simultaneous, and their etiology was at that time puzzling and a subject for the most thoughtful speculation. It was an acute infection of the central nervous system coincident with the epidemic of influenza, but was it a new disease entity or the specific selective action of the influenza toxin on the posterior roots? A noticeable feature of the present recurrence of influenza has been the frequency of fleeting and often sharp, severe, localized types of neuralgic and radicular pains. It seemed, therefore, that this might be considered an influenza complication in spite of a negative history in each case. However, as deviations from this type appeared, the condition gradually simplified itself, and it is now quite certain that this represents only another of the many forms of epidemic encephalitis.

TYPES OF EPIDEMIC ENCEPHALITIS THAT HAVE INCLUDED A RADICULAR SYNDROME

In the preceding part of this paper I have assumed that certain cases of descending radiculitis were closely allied to, and were probably a posterior poliomyelitic form of, epidemic encephalitis. There are certain features about these cases that support this hypothesis. All the patients at one time showed, in the course of a mild febrile disease, a rather definite change in the psyche. This first manifested itself by an acute hallucinatory delirium with confusion and some disorientation, and when this condition gradually cleared up a subdued, apathetic, but not at any time a true lethargic, state remained. Three of the patients showed a decided lack of facial expression, and the tremor, though not of the "pill rolling type," nevertheless was of a coarse, oscillating character, the whole suggesting the "parkinsonian" picture. These symptoms being characteristic of certain cases of lethargic encephalitis, we may venture to suspect a close relationship on the basis of these cases alone.

Soon after observing these cases of radiculitis with delirium, another case was seen which presented the same clinical syndrome. The symptoms began ten days after defervescence from an influenzal pneumonia with lancinating pains in the arms, paresthesia and hyperesthesia with spasmodic twitchings and delirium. The symptoms were in every particular the same as observed in the other cases, and they progressed downward after one week in the arms, gradually giving girdle sensations about the abdomen with spasms of the recti muscles. On the ninth day, the patient complained of diplopia and somnolence, and developed sluggish pupils, bilateral ptosis, weakness of the left sixth nerve; in fact, all the classic features of an epidemic encephalitis. These symptoms progressed, and four days later death resulted (see Case 6). This case was of unusual interest, as the patient presented a definite history of an influenza with a pneumococcus group 1 pneumonia progressing to cure with development of radicular cord symptoms, and then succumbed to a typical acute epidemic encephalitis, the whole forming an apparently unmistakable link between the two conditions.

Another case that also showed this relationship ran a course of the typical radicular syndrome with delirium during the first week. The disease started in the arms, progressed in a descending course, and at the same time the patient complained of diplopia and blurring vision. There was some tendency to lethargy, while a lack of facial expression was noted by her physician. The radiculitis progressed as far downward as the inguinal region and then stopped. The diplopia remained until four weeks after, when I saw her with a left sixth palsy, slight bilateral ptosis, mask-like face, and coarse tremor of hands and feet. We have here again a case with a descending radiculitis which presents evidence of involvement of the midbrain and the nucleus lentiformis (globus pallidus), as well as the posterior spinal ganglions (see Case 8).

One other case (7) which I report in detail shows a similar antecedent descending radiculitis followed by a typical encephalitis.

Case 6.—D. C. D., housewife, formerly a nurse, was seen on Feb. 6, 1920.

Family History.—This was negative.

Past History.—She had one boy, 2 years old. The patient had been well, was six and a half months' pregnant, and had just finished nursing her husband through a mild attack of influenza when she developed it.

Present Illness.—Her illness started with influenza on January 19. Three days later she developed signs of bronchopneumonia, which turned out to be caused by type 1 pneumococcus. Serum was given with a rapid defervescence. and she was afebrile for about ten days, when on February 1 she complained of a "thread-like" feeling in the left side of the neck. This developed in twenty-four hours into a severe knife-like, shooting pain down the arms and back of the neck with such intense hyperesthesia that she could not rest on the left shoulder, nor could she bear the touch of her negligeé. Paresthesia of the arms and hands was present and spasmodic twitching of the arms, beginning in the left, and progressing to the right. The pain was so severe that no relief or rest was obtained without morphin. Forty-eight hours before I saw her she became slightly delirious; the delirium became more and more pronounced and she became somewhat excited, had hallucinations and tried to get out of bed, but she could talk quite rationally when aroused and was oriented. The following day when I saw her the patient was clear as to all the details of her long illness, but when left alone she mumbled and talked to herself, and attempted to get up. She was comfortable, only complaining of an occasional twinge of pain down her arms and a feeling as though "she had had her fingers in lime and soda," with some numbness.

Physical Examination.—This revealed that heart and lungs were normal; the face was bright; the pupils were normal; there were no cranial nerve signs and no tremor except a rare twitching of the arm. Triceps, biceps and wrist reflexes were exaggerated. Leg reflexes were normal. Sensorium: Moderate hyperesthesia over right arm; none on left; no other disturbances. Uterus was enlarged above the umbilicus; the bladder was distended. Temperature for the past week varied between 99 and 100.3 degrees. The pulse rate was 80.

Course of Disease.—February 7: She had complained of some radiating pain around the chest and on the next day about the upper abdomen, asking her husband to unfasten the night sac in the back, as it was too tight. Diplopia was noticed on that day for the first time, and objects seemed blurred. The temperature had been between 101 and 103.8 degrees. February 9: She was very stuporous and apathetic, and slept a great deal. She was definitely lethargic, but could with ease be aroused to an intelligent conversation. Twitchings of the arm and abdomen were present, and some subsultus. Catheterization had been necessary.

Physical Examination.—The patient was muttering to herself; the mental condition has been described above. There were no stiffness of neck or Kernig's sign. There was slight weakness of left sixth nerve, with lack of coordination of eye movements. The pupils were small, equal, irregular and reacted sluggishly to light. Fundi: Veins engorged, outline of nasal borders of both disks was obscured. All deep reflexes were exaggerated; there were no pathologic signs. The pulse rate was from 120 to 130; respiration, 38. Spinal puncture: pressure slightly diminished; cells, 16—all lymphocytes; globulin +. A diagnosis of epidemic encephalitis was unhesitatingly made.

February 10: The condition was more serious. The temperature was between 103.4 and 104.2 degrees; pulse, 150. The mental state was more profound, but she could be aroused. There was tremor of the arms coarse in character and there was an unquestionable lack of facial expression and a bilateral ptosis. She complained that "her neck felt loose," and she had little control over it. There was nystagmus; no other cranial nerves were involved. Distention was troublesome. The fetal heart was still heard.

February 11: The patient's condition was similar to that of the day before; she was more stuporous, but could be aroused. The temperature was around 103 degrees.

February 12: The patient was becoming progressively worse, but could still be aroused to conversation. The voice was hoarse, and there was some difficulty in swallowing. The tongue deviated to the left. She was very somnolent. The temperature was between 104 and 105 degrees.

February 13: The patient died.

This case is of unusual interest and is without doubt the most important in the series. This patient, with a definite history of influenza, developed after defervescence from a pneumonia the syndrome of descending radiculitis, beginning in the highest cervical segment, and progressing downward in the usual fashion as far only as the abdomen. Then followed an oculomotor disturbance and typical symptoms and signs of an epidemic encephalitis with fatal termination. The linking together of the influenza, radiculitis and encephalitis in this one patient suggests a helpful if not conclusive clue as to their relationship.

Case 7.—H. P., a woman, aged 38, married, a clerk, was seen on Feb. 12, 1920.

Family and Past History.—This contained no diseases bearing on the present condition.

Present Illness.—The onset was sudden. While at work on January 12 she developed a cold in the head, with coryza, headache and slight fever. The pains in the head were localized occipitally and became rapidly worse, radiating to the base of the skull and neck. She came home, drank considerable whisky and took quinin. The pain increased in intensity and spread to the shoulders and down the arms to the hands and fingers. The pain was intermittent and lancinating in type, gradually progressing downward, radiating around the chest and abdomen and then to the groin. Here the process stopped and the pain spread more or less upward again, the pain in the occiput reappearing. There was some paresthesia and a hyperesthesia accompanied the progress of the pain. Twitching movements of the muscles were not a feature, but were noted. Delirium was not marked, but was present to a mild degree, accompanied by some confusion. Diplopia was complained of one week after the onset of her illness.

Physical Examination.—This was made by her physician ten days after the onset. The patient appeared to have received a considerable amount of opiates; she was confused and mildly delirious, the pain at times causing her to jump out of her chair. The expression of the face was mask-like. Slight ptosis of right lid and external strabismus in right eye were present. Pupils: The right was smaller than the left and reacted sluggishly to light. Movement of the neck was somewhat painful; the patient held it forward. Heart, lungs and abdomen were negative. The temperature was 101 F., pulse 100. The blood pressure was: systolic, 130; diastolic, 75. The deep reflexes in the extremities were exaggerated, suggestive of the Kernig sign. There was slight hyperesthesia to pin prick of the glove type.

Course of Disease.—The course from then on until about February 1 was a gradual abatement of the symptoms, the temperature ranging from 100 to 102 degrees, reaching a normal level one week before I saw her. Pains became less and less frequent and severe, and the hyperesthesia abated gradually until about February 1 it suddenly became more severe, involving the entire body from neck to groin, being an intense burning pain, so that the rubbing of her clothes was almost unbearable. Asthenia had been marked, and a progressive weakness of the muscles of the arms and trunk had been noted. She had slight pain in her shoulders, chest and groin. Diplopia was still present. Physical examination on February 12 revealed a lack of emotional expression in her face; all movements were slow; slight ptosis; pupils sluggish in reaction, and an external strabismus. There was no deviation of the tongue or facial palsies. Movement of the head on the neck caused slight pain. There was no Romberg sign or gait disturbance. There was atrophy of all the spinal muscles above the waist, sternomastoids, trapezius, muscles of arm, forearm, and hand, and of the chest and abdomen, with corresponding weakness. Reflexes: Triceps, biceps, wrist jerks, knee and ankle jerks were exaggerated. No Klippel Weil. Abdominals present. There were no clonus or extensor responses. There was an occasional twitching of the muscles and a coarse tremor of the arms.

Sensorium: Above Poupart's ligament and below the thyroid cartilage over both anterior and posterior surfaces of the body there was an intense superficial hyperethesia to all forms of sensation. This includes the segmental zones supplied by the C3 to T12. Over the arms in patchy areas was a slight diminution to deep sensation of touch (pressure) and pain. Her skin was dry and the blood pressure was low.

Laboratory Findings.—January 24: Spinal puncture revealed: cells, 84: globulin +. The Wassermann test was negative for the blood and spinal fluid.

February 2: The spinal fluid showed: diminished pressure; cells, 20; lymphocytes, 100 per cent. The globulin was faintly positive; the urine was negative; white blood cells, 11,000; polymorphonculears, 80 per cent.; lymphocytes, 20 per cent.

The progress of this patient has been slow and another report will have to be made at a later date. Improvement has not again been impeded.

There are several unusual features in this case — a typical course of a descending radiculitis progressing downward as far as the last thoracic segment. Here the process reversed itself, a diplopia developed and the symptoms ascended to the occiput with hyperesthesia as the most prominent symptom. The progressive muscular atrophy, while noted to a mild degree in Cases 1, 2 and 4, was marked in this patient. We have, therefore, to deal with a lesion extensive in area — an encephalomyeloradiculitis.

Case 8.—F. S., aged 38, an exterminator, was admitted to medical A service, St. Luke's Hospital Feb. 9, 1920.

Symptoms.—The patient complained of fever, pains in body, twitching of extremities and delirium. The history was obtained from his mother.

Past History.—This was negative.

Present Illness.—He had been suffering with a badly infected tooth for three months. On January 23, after eating four portions of charlotte russe, he had a severe vomiting attack, and also complained of soreness in the tooth, which caused the throat to be inflamed. On January 27, he first noticed sharp, darting pains in the back of the neck behind his left ear, soon traveling to the other side and to the shoulder, and a "pins and needles" sensation in the hands. Fever of 102 degrees was noted by the physician. On January 29 he complained of diplopia and blurring vision which remained until admission to the hospital. On this day the pain was severe and knife-like down the arms and in the back of the neck, and when his mother touched his hands or arms he complained a great deal of the pain produced. This hyperesthesia also involved the shoulder and back of the neck. Numbness and tingling in the arms and hands were noticeable. A spasmodic involuntary twitching of the hands and arms developed soon after so that his arms were "always moving." Within a few days the pain progressed to the upper chest followed by girdle pains around the abdomen, which were accompanied by spasms of the recti muscles. About four days before admission pains, paresthesia, and twitching progressed downward to the legs, but the pains after the first few days were never severe and while in the legs still persisted above. On February 4 he became actively delirious, talking wildly, seeing things and jumping out of bed. This continued up to and after admission. On February 6 a ptosis of the eyes was noticed by his mother, but there was no somnolence or lethargy; in fact he suffered from insomnia. The temperature was about 102 degrees until admission to the hospital.

Physical Examination.—The patient was acutely ill; actively delirious and trying to get out of bed; subsultus; at times he seemed distinctly lethargic, and could be aroused with difficulty. Eyes: There was a lack of coordination of eye movements but no definite paresis of a muscle. Photophobia was present. The pupils were unequal, contracted and fixed to light. The fundi were normal. Twitching of abdominal muscles and of legs was still present. All reflexes were present and active. There were no sensory disturbances.

Course of Disease.—February 11: The patient was delirious and could not be aroused. The pupils were as noted above and twitchings of the muscles were still present; some subsultus.

February 12: He was unable to open his eyes, but could be aroused. Twitchings were less marked.

February 13: Some twitchings were present and there were coarse tremors of the arms. Slight catatonia was noted.

February 14: The patient was not easily aroused, and had some resistance to passive motion; parkinsonian facies were suggested by his appearance.

February 17: He replied somewhat to questions. He had a delirium of a muttering type, but was more quiet and lethargic, at times somnolent. The pupils reacted to light; the right pupil was smaller than the left.

February 26: During the past week there had been slow improvement. His mental condition was clearing and he fed himself. There was less ptosis.

The temperature during the first week after admission varied between 103 and 101 degrees. During the second week it varied between 102 and 101 degrees; in the third week it was between 101 and 100 degrees. After that gradually approached normal.

Spinal fluid examinations showed cell counts of 30, 51, 120, 46, 40 and 30, the cells all being lymphocytes. Globulin was present in excess. The colloidal gold curve was: 1111.51.510000.

The blood and spinal fluid Wassermann tests were negative,

The blood count was: red blood cells, 5,500,000; hemoglobin, 100 per cent.; the white blood cells ranged from 12,000 to 16,000, and the polymorphonuclears from 74 to 86 per cent.

This case shows the syndrome of a descending radiculitis which became attenuated in its intensity as it progressed downward, but presented characteristic symptoms in the legs in conjunction with a diplopia, mask-like face, active delirium passing into a somnolent condition, a combination of radicular, midbrain, basal ganglions, and cortical lesions all progressing toward recovery.

LITERATURE ON RADICULAR SYMPTOMS IN EPIDEMIC ENCEPHALITIS

In the hundreds of case reports with which the literature on encephalitis abounds, it is of interest to note the absence of any that correspond to the cases which have appeared this winter. An occasional patient is reported to have had vague neuritis pains, but not one has had the orderly sequence of symptoms which these have presented.

In a number of excellent case reports on the disease, Barker, Ross and Irwin[3] give one which proceeded as follows: backache, hiccough, pain around the waist with a feeling of constriction, stabbing pain down to the groin and later lancinating pains down the legs. The patient was drowsy, but had no diplopia. The pupils were sluggish. The spinal fluid revealed a pleocytosis, 14 to 48 cells, and an increase in globulin. This case was considered by them to be a meningoradiculomyelo-encephalitis, and it progressed to recovery.

Tilney and Riley,[4] in their nine subgroups of classification of epidemic encephalitis, name a posterior poliomyelitic type and cite a case which presented lethargy, diplopia, paresthesia, pain, a zoster-like eruption over the left trigeminal distribution, and then paresthesia and intense hyperesthesia over sacral second, third and fourth segments of the right leg. The process involved the dorsal root ganglion in the sacral region and the gasserian ganglion as well as the midbrain and cerebrum.

C. DeFano[5] reports a patient who developed severe trigeminal pain in the course of an epidemic encephalitis, and Buzzard[6] one of a pregnant woman with onset of acute pain down the left arm followed by spasmodic twitchings of the arms, diplopia of transient character and delirium. The differential diagnosis from chorea gravidarum was difficult, but he considered it an abortive case of encephalitis.

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[3] Barker L. F.; Cross, E. S., and Irwin, S. V.: On the Epidemic of Acute and Subacute Inflammation of the Nervous System Prevalent in the United States in 1918-19: Encephalitis, Encephalomyelitis; Polyneuritis, and Meningo-Encephalo-Myeloneuritis, Part 1, Am. J. Med Sc. **159**:157, 1920.

[4] Tilney, F., and Riley, H. A.: Epidemic Encephalitis, Neurological Bull. **2**: 106 (March) 1919.

[5] DeFano, C., and Ingleby, H.: Preparations for Cases of Encephalitis Lethargica, Proc. Roy. Soc. Med., Section of Pathol. **12**:42, 1919.

[6] Buzzard: Encephalitis Lethargica, Proc. Roy. Soc. Med., Section of Neurol. **12**:56, 1919.

McNalty,[7] in his report on the epidemic in England in 1918, noted the following facts: Pains were not of infrequent occurrence, either diffuse all over the body or localized to particular regions, such as the neck, back or limbs. Twitchings occurred in the face and limbs. Hyperesthesia of the skin was noted especially in the limbs and soles of the feet. He mentioned five patients who had paresthesia and "neuritis" in the arms and legs.

It can be seen from these references that involvement of the posterior roots is not a new occurrence with the epidemic this winter, but there have been as yet no reports of a descending radiculitis with delirium appearing as a form of encephalitis.

PATHOGENESIS

There is definite clinical evidence from the case reports given in this article that an involvement of the posterior spinal roots is of frequent occurrence, appearing either alone or in conjunction with signs of an encephalitis. From the pathologic reports it is difficult to evaluate the extent of the lesions, for although observations on the brain are legion and reveal that any part may be affected, those on the spinal cord are few and inconclusive. As all my case reports concerned persons who had recovered, except in one instance in which no necropsy was permitted, I have been able to draw conclusions only from two reports. Strauss and Loewe [8] have noted a round cell infiltration in the posterior root ganglions. At the Rockefeller Institute Flexner and Amoss [9] have noted a like involvement with some small hemorrhages and perivascular infiltration similar to that noted in the cerebrum. With these few pathologic reports, and from the nature of the symptoms and physical signs in this spinal form of encephalitis, it seems quite certain that the lesion is located in the posterior spinal roots and possibly in the parenchyma of the posterior root zone of the spinal cord. There is some mild meningeal inflammation, and the presence of delirium indicates an involvement of the encephalon.

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7. McNalty: British Local Government Board, Report on Encephalitis Lethargica, N. S., 1918, No. 121.

8. Strauss, I., and Loewe, L.: The Etiology of Acute Epidemic Encephalitis. Presented before the New York Pathological Society, Feb. 11, 1920.

9. Personal communication from Dr. H. L. Amoss.

CONCLUSIONS

As one begins to consider the manifestations that epidemic encephalitis has shown clinically and pathologically, it becomes clear that this disease may invade almost any portion of the nervous system. It has a predilection for the basal ganglions, nuclei of cranial nerves, and, as we see from the case reports in this paper, for the posterior roots, but other parts are not exempt from its invasion.

Acute descending radiculitis is an infection of the posterior spinal roots which may appear as a separate clinical entity and pursue a most stereotyped course, eventually ending in recovery. It may also antecede, by several weeks or days, in a more or less typical but usually attenuated course, the cerebral form of epidemic encephalitis. Assuming that a posterior root syndrome may be a prominent complication of epidemic encephalitis, such symptoms as myoclonic twitchings, hyperesthesia and radicular pains may be considered as much a part of the picture in the disease as diplopia, somnolence and cranial nerve palsies.

The etiology of this disease has required considerable study. Its similarity pathologically to poliomyelitis has been noted, but as encephalitis becomes more familiar to us, this resemblance soon diminishes, and we appreciate that it has many different manifestations. It is not within the scope of this paper to discuss the history of the disease except to note that it has appeared in a similar form coincident with previous epidemics of influenza. Its occurrence in epidemic form in the winter of 1918-1919 and again in 1919-1920, with sporadic cases occurring between the two epidemics, seems to link it closely to influenza. Efforts to determine the etiologic organism in influenza have not been too decisive, the only bacteria found with frequency having occurred in its complications, most probably symbiotic with the yet undiscovered micro-organism which causes the disease. A similar uncertainty has obtained in encephalitis, for a number of investigators have reported the presence of an organism, among them Strauss and Loewe,[8] who reported a "globoid" organism a few months ago. The question still appears to be unsettled.

Reports scattered through the literature on the previous epidemics of influenza comment on its predilection for nervous tissue, and Menninger,[10] writing of the 1918 epidemic, remarks that "the quantitative specificity of the influenza neurotoxin is confirmed by its remarkable potency and versatility; the large number and wide variety of psychic and encephalopathic lesions produced being one of the most striking neuro-psychiatric features of the disease."

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[10] Menninger, K. A.: Psychoses Associated with Influenza, Arch. Neurol. & Psychiat. **2**:291 (Sept.) 1919.

In a previous section of this paper was mentioned the frequency with which pains have occurred this winter as sequelae to, or independent of, an acute influenza. These have been most commonly intercostal or around the abdomen, but have also been localized in the extremities. They have been radicular in their distribution and of a neuralgic root type, sometimes lasting with great intensity for only twenty-four hours, and again resisting all efforts at relief and persisting several weeks. Hyperesthesia over the dermatomes was present in many cases. It seems that the close relationship between these radicular pains and influenza, and their similarity to the radicular types of epidemic encephalitis may suggest a possible, but of course as yet unproved, common etiology.

The author begs to acknowledge with great pleasure and thanks the privilege of observing and reporting the material in this paper in Cases 1 and 8 to Dr. Austin W. Hollis, Case 2 to Dr. F. Warner Bishop, Case 5 to Dr. W. H. Glafke, Case 6 to Dr. Kyle Steele, and Case 7 to Dr. Waldo B. Farnum.

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ENCEPHALITIS WITH INVOLUNTARY MOVEMENTS

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The four cases herein reported show such a characteristic picture that they may be classed as belonging to a somewhat individual type of encephalitis. In fact, after two of them had been described,[1] an article [2] came to the writer's attention containing a clinical picture differing in minor particulars only from that of the cases I report.

Few cases of encephalitis with involuntary movements have been described. In those reported by Buzzard,[3] the movements seem to have differed in several interesting ways from those of my patients. He states that "all these movements cease during sleep," whereas in the three cases that I observed they were much more active during sleep. Buzzard lays no emphasis on the rhythmic character of the movements — one of the most striking features in my cases. Only in Cases 10 and 12 does he speak of movements of the shoulder girdle muscles; I was particularly impressed with the bilateral and symmetrical distribution of the involved muscles and that they were especially those of the neck and shoulder girdle. In the article by Sicard and Kudelski no mention is made of the effect of sleep on the movements or of bilaterality. They give an unfavorable prognosis; whereas the four patients I described have all recovered from the acute stage and seem to be improving with respect to the involuntary movements. Bladder disturbance has been a prominent symptom and in each case the cerebrospinal fluid has shown evidence of inflammation; Sicard and Kudelski say the sphincters are unaffected and the spinal fluid normal.

REPORT OF CASES

CASE 1.—*History.*—S. H., a boy, aged 11, entered Wesley Hospital Nov. 30, 1919. Ten days before entrance he complained of a sticking or prickling sensation in the right side of the face. Two or three days later he began to vomit. On the day of entrance fever was first determined, and about 5 a. m. the boy had a convulsion; convulsions recurred during the day.

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[1] Med. Clin. North America **4** (May) 1920.

[2] Sicard and Kudelski: Encephalites Aigues Myoclonique, Bull. et mém. Soc. méd. d. hôp. de Par. **44**:95 (Jan. 29) 1920.

[3] Encephalitis Lethargica, Proc. Royal Soc. of Med., Neurol. Sec. **12**:56 (Aug.) 1919.

When first seen at the hospital he was at times rational, and complained of pain in the right side of the face; at other times he was delirious, shrieking and yelling. When I first saw him his posture was highly characteristic of meningeal irritation. The boy was lying on his side, the head was drawn back, the knees and hips sharply flexed.

Examination.—There was no opposition to passive movements, except slight rigidity of the neck, and no Kernig's sign. The pupils were dilated, reacting slightly, if at all, to light; ocular movements were normal. The reflexes could not be obtained because of the incessant movements of the patient. He had the appearance of being toxic. There was no distinct paralysis. It was impossible to test sensation. The spinal fluid was under increased pressure and showed about 1,500 cells to the cubic millimeter.

Course of Disease.—During the following six weeks the patient continued in a more or less irrational condition; he frequently involuntarily discharged urine and feces; he had no distinct convulsions. About two weeks after entrance he had to be catheterized. During six weeks the white blood count varied between 22,000 and 16,000, the temperature between 104 and 100 F., the pulse between 150 and 110. The urine was negative. On January 12 he appeared much brighter. Notes made at that time state: "There is more or less persistent, interrupted trismus; a slight spasm in the temporals and masseters, occurring about two or three to the five second period, showing no distinct regularity. There are also frequent clonic spasms of the thoracic and abdominal muscles and the diaphragm. These spasms seem to overflow into the extremities where, however, they are not so prominent. The voice is that of a patient with well developed general paresis; it has the same monotonous, slightly nasal quality, is tremulous, in the regular paretic tempo, and the syllables are not cleanly cut, mistakes in enunciation occurring in complex words. All voluntary movements are possible. The plantar reflexes are much diminished but flexor in type. The right ankle-jerk is normal, the left is absent. The right knee-jerk is normal, the left diminished. It is impossible to demonstrate the abdominals and cremasterics because of the constant involuntary twitchings. He says that a pin prick on either leg feels like cold water. It is not felt at all on the anterior surface of the thighs. There is no Kernig sign, but slight rigidity of the neck which seems almost volitional; he holds his neck rigid, then suddenly gives up and allows the chin to flex practically to the chest. The mental confusion has entirely disappeared and he can control bowels and bladder." Lumbar puncture at this time revealed no increase in pressure; the Ross-Janes test was positive; 20 cells to the cubic millimeter.

On February 10 he felt quite well and had been up and about for a week. Notes made at this time state: "The patient does not feel any involuntary movements. On examination, however, a peculiar, complex spasm or sudden contraction of the pectorals, sternocleidomastoids and diaphragm, is seen to occur about forty to fifty times a minute. It is impossible to determine whether there is a relation between the movement and respiration. He is able, for example, to take five deep inspirations without the spasms interfering with the rhythm; they do, however, interfere tremendously with ordinary quiet breathing. Associated with the movements of the above mentioned muscles there are spasms of the latissimi dorsi, the left teres group, both biceps — more spasms of the left than of the right — and the pronator and flexor groups of the left hand. At times the adductors of the left thigh twitch with the above mentioned muscles; also there are irregular spasmodic twitchings of the recti abdominis, at times together, at other times separately. A convulsive twitch

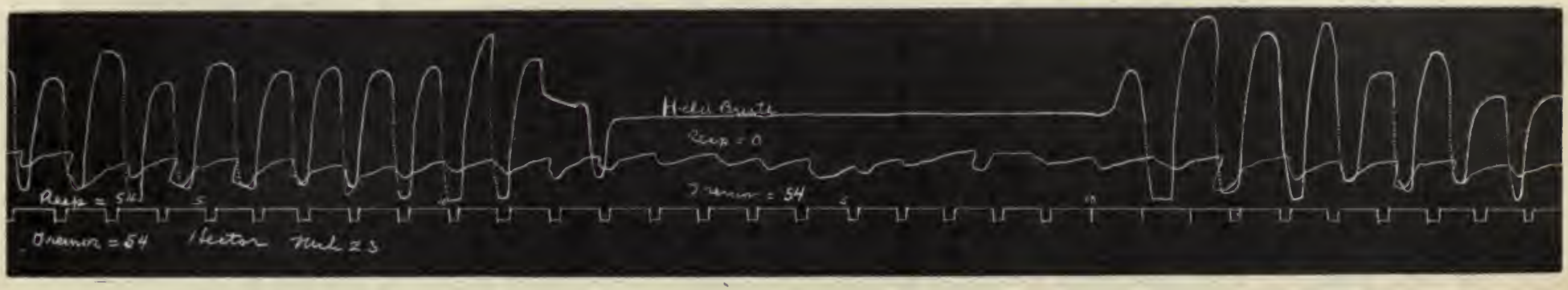

Fig. 1 (Case 1).—The line with the large excursions represents the respiration. The more irregular line of smaller excursions represents the muscular twitch. The bottom line represents the time. The synchrony of the muscular twitch and the respiration is quite obvious. For a period of ten seconds the patient held his breath. The tremor went on with an altered rhythm.

elevates the right eyebrow and retracts the right angulus oris. This is irregular in tempo, is markedly different in intensity at different moments, and occurs about eighty times to the minute. At times the movement around the eye is more marked, at others the movement around the mouth. There is tremor of the tongue, both of the trombone type and a finer tremor along the edges. No disturbance of sensation is demonstrable. Plantar reflexes are normal, ankle and knee jerks are present, greater in the right side than in the left; the abdominals are diminished, deep reflexes in the arm are present on the right side, questionable on the left. Pupils and ocular movements are normal."

On April 15, the patient was observed during sleep. The twitch of the sternocleidomastoids, the trapezei and the pectorals was more marked than during his waking hours. Fluoroscopic examination showed a twitch of the diaphragm synchronous with the twitch of the shoulder girdle muscles.

Case 2.—*History.*—T. A., a man of 45 years, was seen with Dr. A. J. Hedgcock at Decatur, Ill., on Feb. 1, 1920. I am indebted to Dr. Hedgcock for notes on the case. With the exception of a nervous breakdown five years previously, family and personal histories were negative.

While driving on the afternoon of Jan. 18, 1920, a "terrific pain" suddenly developed in the right arm. The pain kept him awake all night, and the following day it extended across the back into the left arm and up the back of the neck.

Course and Treatment.—When first seen, January 20, the patient complained of severe pain down the right arm. His temperature was 99.5 F.; the findings otherwise were negative. On January 21 the pain involved both arms and a tendency toward myoclonia was noted. The pain at this time was so intense that a hypodermic injection was necessary. The following day there was slight delirium, the myoclonia had become intense and extended to the abdominal muscles. The delirium increased, the temperature was 100 F., the pulse 90, and respirations 20. The Wassermann reaction with blood and spinal fluid was negative. There were 6 cells to the cubic millimeter of spinal fluid; the globulin test was negative. The blood showed 10,500 white cells, of which 85 per cent. were polymorphonuclears. The urine contained a trace of albumin and a few hyaline casts. Three days later pulmonary edema developed. Oxygen was given the entire night with cardiac stimulants and at 7 the next morning the patient was much better. Respirations at this time were unequal and shallow, giving the appearance of diaphragmatic involvement.

The patient was seen by me about this time. There was slight drooping of the right eyelid and weakness of the right arm. Marked twitching of the deltoids, pectorals and abdominal muscles was present. Pupillary reflexes were normal, the tendon reflexes were diminished, the Babinski sign was absent. The spinal fluid contained 10 cells to the cubic millimeter; the Nonne reaction was faintly positive. Leukocytes had risen to 15,000, of which 88 per cent. were polymorphonuclears. The delirium was of the toxic type, strongly suggesting that of alcohol.

About a week later the patient had to be catheterized, the only time involvement of the bladder was noted.

For the next few weeks a condition of toxemia continued, twice interrupted by consolidation of the lung, once on the left side and once on the right, probably hypostatic. There was gradual lysis, the delirium faded, and the only defect remaining was a gradually improving paralysis of the right arm and the twitching of the recti abdominis.

Case 3.—*History.*—A woman, 20 years old, whose family and previous history were unimportant, Jan. 16, 1920, had a stiff neck and a severe, sharp pain running from the left side of the neck down into the shoulders and to the heart region which in the course of a few days extended down the left side to the foot. At the same time tremor of the hands and arms developed which gradually increased in intensity until, after a few days, it entirely prevented the patient from feeding herself or even raising a glass to her lips. During this time neither headache, rigors, chilliness nor flushes indicated fever. Shortly after the onset she began to have what were described as "frightened spells," with screaming. These spells occurred with considerable frequency, more often at night and apparently more often when, for any reason, she was emotionally aroused. There was also much delirious talking, especially at night.

There had been no difficulty with vision and no disturbance of speech. For three days previous to examination the urine had been hard to start, even when the desire was great.

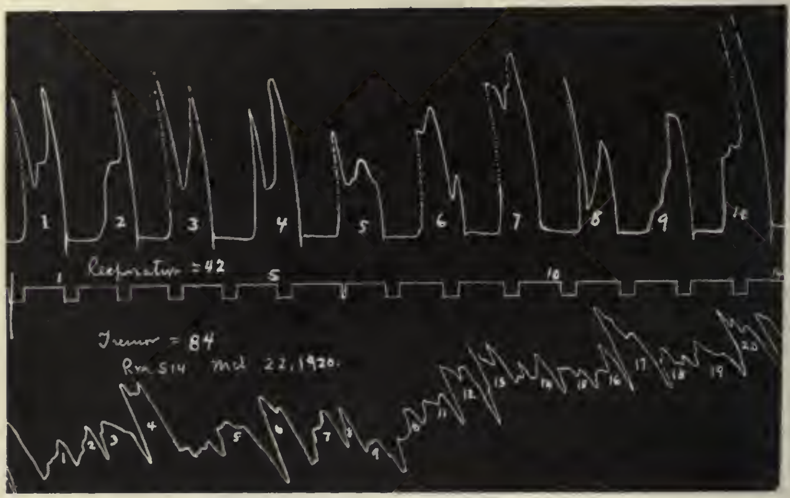

Fig. 2 (Case 3).—The upper line charts the respiration, the middle the time marker and the lower the tremor. A definite relationship seems to exist between the twitching and respiration; there are two of the former to each one of the latter. A comparison of the respiratory tracings in Figures 1 and 2 is interesting, in that Figure 1 shows a single wave with an unbroken summit, whereas Figure 2 shows a double summit with a deep valley between. This valley was probably caused by the muscular relaxation occurring between the two twitchings. In both these cases the diaphragm was seen to twitch synchronously with the main twitching of the skeletal muscles.

Examination.—The patient was first seen on Feb. 23, 1920, at which time the following notes were made: "The patient is a thin, sallow girl, with a peculiar rash over the shoulders, chest and buttocks. She is in a profuse perspiration and obviously much disturbed by the trip she has just made to the hospital. The rash is petechial in appearance and yet on areas of the skin where there are hairs it seems to be a fine folliculitis. This impression is supported by the fact that her skin shows evidence of neglect.

There is frequent, fairly rhythmic twitching of the adductors of the arms; with each twitch there is an inspiratory movement which seems to be due to movement of the ribs. With this movement the recti abdominis are stretched upward; the umbilicus moving upward about an inch; there is no apparent movement of the diaphragm. Synchronous with this spasmodic twitching there is a twitching of the muscles of the left leg, especially the quadriceps femoris and the extensors of the toes. There is also a slight contraction of the flexors of the forearm and the pronators that occurs simultaneously with the twitching of the shoulder girdle.

Ocular movements and pupils are normal, the right optic disk is redder than the left. The left side of the face moves a trifle more than the right on volition and in emotional expression. There is no resistance to passive movements except slight rigidity of the neck.

The biceps jerks are normal, triceps and wrist jerks are absent, the right abdominal reflex is normal, the left absent. The presence of knee jerks and ankle jerks is questionable; probably those on the left are absent, and those on the right much diminished. The plantar reflex on the right is normal; on the left it is either absent or slightly in extension. The patient states that pin prick does not feel nearly as sharp on the left side, especially the left leg, as on the right. The ethyl chlorid spray is felt as distinctly colder on the right side. It is difficult to demonstrate any change in heat sensation. Joint and tendon sense apparently is normal. Roughly examined, the visual fields are normal.

With the major or chief twitch or spasm involving the respiratory muscles and, to a less extent, those of the arm and leg, is an almost constant tremor. This tremor is especially marked with active movements which, however, diminish the intensity of the chief twitch. During ordinary quiet respiration the twitch, occurring as it does about fifty times to the minute, seems to be the only respiratory act. However, when forced and deep respiration is attempted the flow of air is steady, it is not interrupted, and the muscular twitch, as for example that of the pectorals, is distinctly diminished. Ordinarily quiet breathing is superficial, determined by the spasmodic twitch of the respiratory muscles. The involuntary movements are all increased during sleep.

The patient is well oriented and her memory is good. As soon as allowed, however, she relapses into a more or less delirious condition in which she babbles unintelligibly and makes purposeless movements with the hands.

During the examination she had a "frightened spell." A look of terror and suffering appeared on her face, and she cried out "Oh, my leg, my leg, it's so cold." There were no special changes in the eyes, the body began to twitch rapidly; the left leg was partly flexed and held more or less rigid; there was a tendency to assume a position similar to the opisthotonus of hysteria. She reached for her mother, who was out of reach, then for the examiner. Respiration was rapid and a profuse perspiration appeared which soaked the sheet. The entire spell lasted about forty-five seconds without loss of consciousness.

White counts on the 24th and 25th were 14,700 and 17,000, respectively. The temperature ranged between 99.6 and 100.4 F., per rectum.

Pressure of spinal fluid was not increased; serum-albumin was increased; the Ross-Jones reaction was positive; there was no increase of cells.

Case 4.—*History.*—A man, 48 years of age, was seen with Dr. Magnuson at Wesley Hospital, March 18, 1920. About Jan. 1, 1920, while at work in a coal mine he had been struck on the top of the head by a rock, about the size of a fist, falling two or three feet. He was not knocked down, but a swelling about half the size of a hen's egg appeared on the top of his head.[4] He worked on throughout the day and for the next three days. On the night of the fourth day a severe lancinating pain was felt just to the left of the midvertex. It woke him out of a sound sleep, and he was awake the rest of the night. The pain prevented sleep for the next three days and he thought it "would drive him crazy." At the end of this time he became amnesic; at least, he said that for three days he suffered pain and then lost consciousness. He knew nothing of the next three weeks, two of which were spent in a hospital, the last at home. He had been told that he acted naturally most of the time, but occasionally would talk "funny." He was in bed until March 5 because of weakness and giddiness.

At the time of entrance to the hospital he complained of weakness, lightheadedness and occasional throbbing pain through the stomach toward the left side of the abdomen.

Examination.—The patient was a large man with good color, but apparently far below his ordinary weight. The expression was rather "anxious and strained." He walked with the feet well apart, his steps were short and careful; there was no distinct ataxia either with open or closed eyes. All voluntary movements were well executed. The cranial nerves (including the optic disks) were normal; the reflexes were normal except that the cremasteric and abdominal reflexes appeared to be a little diminished; sensation was normal. There was frequent twitching of the sternocleidomastoids, cervical erector spinae and both trapezei — the right twitched more than the left. The twitch occurred about once per second. It was practically unaffected by voluntary movements, although when the muscles were actively used the twitch appeared to occur as an epiphenomenon. The patient was unconscious of the occurrence of this movement. Later when observed in sleep the twitching was obviously more active than during the waking period. The urine and blood were normal. The pulse varied between 100 and 80 during the nine days in the hospital. Lumbar puncture was not done. Fluoroscopic examination failed to show any twitching of the diaphragm.

SYMPTOMS IN AUTHOR'S CASES

A few of the details of these cases seem to deserve some additional comment.

Pain.—This was a prominent symptom. In all four cases it was the first symptom and was extremely severe and sharp. In Case 1 it was sufficiently intense to cause the boy to scream in the midst of his delirium. The fourth patient thought it drove him out of his mind into three weeks of delirium. In Case 3 the pain spread from the left shoulder and neck down the left side into the leg. During the one "frightened spell" observed it was complained of, especially in the leg. In Cases 1 and 4 the pain was in the right and left halves of the head,

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[4] Relative to the relation of the trauma to the development of encephalitis, Cleland and Campbell: The Australian Epidemic of Acute Encephalitis, J. Nerv. & Ment. Dis. **51**:143 (Feb.) 1920, assumed that head trauma may act as an agent occasioning the localization of the inflammatory process in the brain.

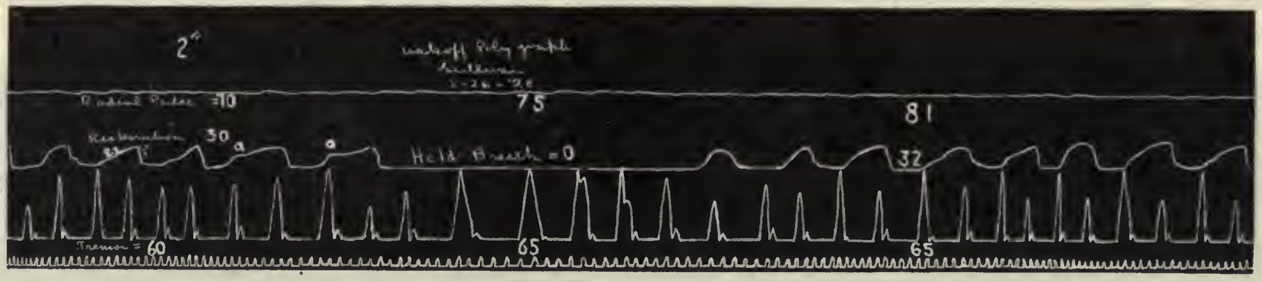

Fig. 3 (Case 4).—The upper line represents the radial pulse; the second line, respiration; the upward stroke, expiration, and the downward stroke, inspiration (see Ex. and In.). The third line represents the twitching, and the bottom line marks the time in seconds. There is a definite ratio of two twitchings to one respiratory cycle; also a slight pause (*a*) of the respiratory lever is recorded occurring midway between the summits of the regular respiratory waves. This was caused by the intervening twitching. The summits are inverted; the twitching of the sternocleidomastoid and the trapezei was sufficient to halt, momentarily, the sinking of the chest walls. There is no fixed relation between the pulse and the twitching.

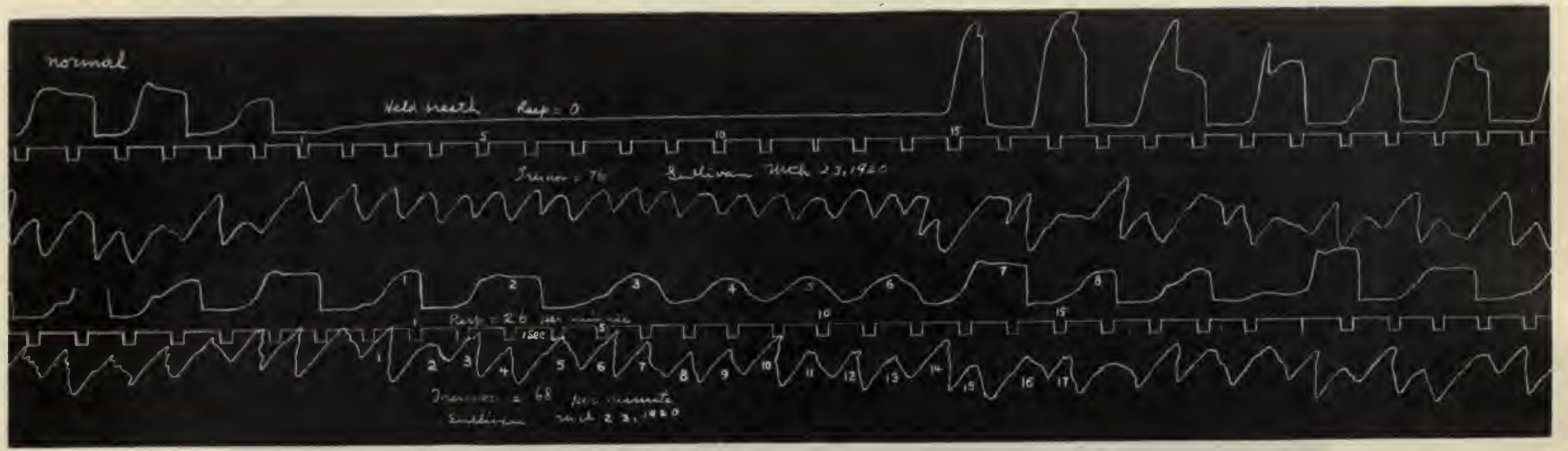

Fig. 4 (Case 4).—The top line represents respiration, the second, time; the third, the tremor; the fourth, respiration; the fifth, time; the sixth, the tremor. In this tracing there is no obvious relation between the twitching and respiration. In this case the muscular movement was much less than in either of the others, and occurred in a man of about 50 years. The apparent regularity of the tremor during the time the patient held his breath (in the upper tracing) is probably due to the fact that the respiration ordinarily moved the lever in an irregular manner. The same number of twitchings are recorded in the ten second period during which he held his breath as during ten seconds of breathing.

respectively; in Case 2 it was first in one shoulder and then in the other. Possibly this pain was due to disease of pain centers in the thalamus, as its character was suggestive of such an origin.

Fever.—In each case fever seemed to be absent for the first two or three days; at least there were no chills, rigors or flushings. The 11-year-old patient developed a temperature of 104 F.; the temperature in the other patients ranged from 100 to 101 F.

Delirium.—The mental symptoms in the three cases closely observed were a toxic delirium with restlessness, an elusive consciousness, which could be held for a few moments at a time when questions were grasped and correctly answered, hallucinations of sight and sound, and in two cases reactions strongly suggestive of terror.

Pulse and Respiration.—In Cases 1 and 3 the rapidity of pulse and respiration was out of proportion to the fever. This disproportion seemed sufficiently definite to be of diagnostic value.

Sweating. — This was especially profuse in Case 3 during the freightened spell. In Case 1 severe sweating was noticed on several occasions.

Bladder Disturbance.—This was present in three of the cases. The symptom has occurred so frequently in cases of encephalitis of this and other types that it is probably of the same nature and origin as the bladder disturbances of pseudobulbar paralysis.

Meningeal Irritation and Spinal Fluids.—In Cases 1 and 3 there was evidence of irritation of the meninges. In three of the cases there were inflammatory changes in the fluid.

Rhythmic Movements.—In the three well observed cases, the patient was quite unconscious of these movements. In Cases 1 and 3 they strongly suggested the movements caused by interruptions of a fairly strong galvanic current. It seemed incredible that the patients were unconscious of them; that they were not continually greatly plagued by them.

In Cases 1, 3 and 4 the movements were decidedly more marked during sleep. Excitement also increased them. Voluntary movement had almost no effect on them. During deep voluntary inspiration the twitches of the sternocleidomastoids could be seen going on in their regular rhythm (see tracings).

From the tracings and the clinical observations, it would appear that the clonic or twitch-like, rhythmic, bilateral movements of muscles used in forced respiration had a definite relation to the respiratory rhythm. The rate of twitching was 54 per second in Case 1, 84 in Case 3 and 60 in Case 4. That would seem too wide a variance if it were not for the fact that the respirations varied from 54 in Case 1 to 42 in Case 2 and 30 in Case 4. In short, the rate of twitching equaled the respiratory rate or was double this rate.

Therefore it seems safe to assume that the twitches are expressions of the involvement of some complex or center in the midbrain or hindbrain that is in direct connection with the respiratory center. An arrangement controlling the muscular phenomena of respiration similar to that controlling walking may be assumed. Thiele [5] concluded from his experiments on dogs that there was a center in the posterior part of each optic thalamus that controlled the coordinated movements of walking.

If it be assumed that walking is controlled from the lower centers in a coordinated manner because of its automatic or habitual character, it seems quite likely that respiration is regulated at as low or even a lower level.

These bilateral movements can scarcely be considered as being of the nature of jacksonian cortical movements. They are too uniformly bilateral and symmetrical. It is highly questionable that cortical movements are ever rhythmic, whereas those movements that, because of their involuntary nature, suggest a subcortical control, are rhythmic. Stewart and Pike have shown the essentially rhythmic action of respiratory centers in the medulla.[6]

I, therefore, assume that the rhythmical movements in these cases are caused by involvement of a mechanism or center in the medulla.

I am indebted to Dr. F. C. Becht of Northwestern University Medical School for a great deal of patience and persistence in securing tracings from Cases 1, 3 and 4.

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[5] Thiele: Efferent Relationship of the Optic Thalamus and Deiter's Nucleus, J. Physiol. **32**:358, 1905.

[6] Stewart: Some Observations on the Behavior of the Automatic Respiratory and Cardiac Mechanisms, Am. J. Physiol. **20**:407, 1907-1908. Stewart and Pike: Further Observations on the Resuscitation of the Respiratory Nervous Mechanism, Am. J. Physiol. **20**:61, 1907-1908.

DIFFERENTIAL DIAGNOSTIC DIFFICULTIES IN PERNICIOUS ANEMIA WITH ASSOCIATED MENTAL DISORDER

REPORT OF A CASE DIAGNOSED FOR SOME TIME AS ARTERIOSCLEROTIC INSANITY *

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Much has been written on pernicious anemia, particularly from the point of view of the internist and from that of the neuropathologist, but with the exception of Barrett's[1] excellent articles and Lurie's[2] more recent communication, relatively little has been contributed in English, at least on the psychiatric aspect of this condition. This is especially true with reference to the difficulties that arise in patients who present arteriosclerotic symptoms with mental features before it can be definitely demonstrated, by neurologic examination or by the blood picture, that the case is either purely one of pernicious anemia with mental symptoms, or a combination of arteriosclerosis and pernicious anemia accompanied by mental disorder. As monographic literature points out, and as hospital experience everywhere daily confirms, pernicious anemia with cord changes is more common than is recognized by the conventional textbooks on nervous and mental diseases. If the cases in general hospitals and in private practice were, or could be, followed closely by the neuropsychiatrist, a greater number of patients showing varying degrees of abnormal psychic reactions would probably be found. This might also be said of arteriosclerosis, and is borne out by the author's observation of cases seen in several outpatient services during the past three years.

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* Read before the March meeting of the Detroit Neurological Society, March 11, 1920.

[1] Barrett, A. M.: Mental Disorders and Cerebral Lesions Associated with Pernicious Anemia, Am. J. Insanity **69**:1063, 1913; Mental Disorders Associated with Pernicious Anemia, Fifth Biennial Rept. State Psychopathic Hospital, Ann Arbor, Mich., biennial period ending June 30, 1916.

[2] Lurie, L. A.: Pernicious Anemia with Mental Symptoms, Arch. Neurol. & Psychiat. **2**:67 (July) 1919.

There are probably many more cases of both arteriosclerosis and pernicious anemia that show associated mental symptoms than is generally recognized. Since the associated mental symptoms are not necessarily characteristic of either of these two diseases, and the blood picture characteristic of pernicious anemia in general, and the neurologic picture in particular — when the cord is involved — may both suddenly appear in cases that have hitherto shown only symptoms that belong to, or might be interpreted as peculiar to, arteriosclerosis, it becomes apparent how important it is to be guarded in any attempt to differentiate arteriosclerotic insanity from pernicious anemia associated with mental disease. It is quite possible that both conditions may exist in a given case. The following case is reported as an example.

REPORT OF A CASE

History.—(Case No. 2586, State Psychopathic Hospital, Ann Arbor, Mich.; courtesy of Dr. A. M. Barrett, director.) S. L. G., a woman, aged 54, married, housewife, an American, was one of nine children. One sister died of pneumonia, and one of cancer, ages not given. One brother died of cerebrospinal meningitis at the age of 16, and another brother who was feebleminded, died at Traverse City (Michigan State Hospital). The family history was reported as otherwise negative.

The patient was bright in school, but somewhat retiring and seclusive in her habits, preferring books and home to outside companionship. She married, happily, at the age of 20. She was a good housewife, but inclined to be overeconomical, disinclined to have a dollar spent unless it was absolutely necessary. Her religious life was not out of the ordinary.

Previous Medical History.—This was reported as negative, except for a brief "nervous spell" twelve years before.

Present Illness.—According to the husband, the present illness was gradual in onset, and extended over a period of at least three years previous to admission to the State Psychopathic Hospital on May 27, 1919. At that time the patient expressed the desire to take care of the chickens in order that she might have the proceeds from that part of the farm business. This idea apparently grew out of the patient's desire to raise sufficient funds to visit a son who was then living in the West. The husband granted her request and agreed to do the more difficult work, but insisted that she look after the poultry. She did so for a year with fair success. Then (two years ago) she began to develop delusions of a whimsical character. Because the hens were laying poorly and the chickens dying off, she imagined her husband was killing them and selling them and the eggs in order to buy dresses and furniture for a woman close by. Later she included every woman of the neighborhood in her delusions but never set traps to catch or expose her husband. Then she imagined everybody was ridiculing her. She began to suffer from sleeplessness and restlessness and lost weight. A year before admission, she began to have hallucinations and grew irritable, and on several occasions threatened her husband. She frequently showed emotional instability, crying most of the time. The husband furnished no history of any similar previous attacks, but spoke vaguely of her suffering a "nervous breakdown" about twelve years before, being confined to bed for about three weeks. After recovering she worried about things in general and did not seem to reach her normal level for almost a year.

Physical Examination.—On admission, the patient appeared to be in a generally rundown condition. The musculature was poorly developed and of poor tone. There was almost no panniculus. The facial expression was one of apprehensiveness. The skin was dry, warm and fairly elastic, but showed a marked pallor. There were no scars, eruptions, or abnormal pigmentations. The neck was negative except for noticeable venous pulsations. The tongue showed only a slight fine tremor. Examination of the cardiovascular system revealed that the apex of the heart was in the fifth interspace at the nipple line. The heart sounds were fairly well heard and the only abnormality was a slight systolic murmur at the apex which was apparently hemic in type. The arteries and veins showed slight, if any, evidence of sclerosis, but there was some varicosity of the veins in the lower extremities. The pulse (rate 100) was of fair quality, rhythmic and synchronous. The blood pressure was systolic, 150; diastolic, 96. A week later, the heart sounds were of poor quality, accentuated in all areas, and the blood pressure was systolic, 160; diastolic, 100. There was a distinct arcus senilis in each eye.

Neurologic Examination.—On admission the neurologic examination was negative except for fine tremors of the extended hands, tongue and closed lids.

Laboratory Examinations.—Urinalysis showed nothing of any significance with the exception of a rather low specific gravity (1.002) and the presence of a few white blood cells.

Blood: Examination of the blood showed a red count of 4,000,000 and a white count of 5,850, a hemoglobin of 75 per cent. and a negative Wassermann test.

Mental Examination.—There was nothing unusual in the patient's general appearance and attitude toward the examiner except for a slight flushing of the face and an inclination to be playfully cynical. At times during her narrative she was slightly emotional. There was no evidence at that time of any abnormal psychomotor activity other than that which appeared to be associated with the slight circulatory disturbance, mild tremors and arrhythmia with inconstant tachycardia.

She denied dreaming, but admitted that she had frequent crying spells which prevented her from going to sleep. Memory for remote events was unimpaired. Simple calculations were done deliberately and slowly. She returned the "Shark Story," however, as follows: "The son of an oriental governor was on a boat when he was struck by a shark. He was cast overboard."

The patient was correctly oriented in all three spheres. Her stream of thought was clear, but not always relevant, and was very circumstantial. She went into endless details about her husband's mistreatment of her in the matter of the chickens, and it was almost impossible to keep her from reverting again and again to this topic. Except for this delusional trend she was logical.

The patient's content of thought has already been referred to in the anamnesis furnishel by her husband and in the preceding paragraph. It is further elaborated in the patient's own narrative, which adds facts of value relative to her physical condition as well as to the nature of her mental disturbance. In addition to the "nervous" condition (twelve years before) mentioned by her husband, she spoke of having had some form of cardiac disturbance four years before admission. She fell to the floor and was unconscious for an hour. A year later she had a similar attack. Both attacks were sudden in onset, and were preceded by a feeling of weakness but not accompanied by vertigo. The account of these attacks was confirmed by her husband. Her mental symptoms developed about that time.

Her narrative, condensed, was as follows: "I was anxious to see my son who was in the West. My husband, feeling that we could not bear the expense of the trip, led me to ask him to turn over the profits of the chickens to me. Mother gave me some chickens as personal property, he took the money from the eggs and pretended to pay the grocery bills with it. When I complained of having no clothes he said I could have all the profit from the chickens. That was three years ago." She then went into endless detail about his deceit, attention to other women, etc., and continued: "About a year ago this grew gradually worse. I became nervous, cried a great deal, and was almost frantic over my losses. Last Christmas he even went so far as to change a rooster on me. One afternoon he took me to town; on our return eleven pullets were gone. Later in January, I received a message from my son in the West that he had influenza. I went to town for the telegram and on my return found more chickens changed and gone. The longer it went on the crazier I became over it."

When questioned closely why her husband did all these things she could offer no other reason than that he was not contented with her. She apparently had no insight into her condition. That her husband had been considerate and patient with her was confirmed later by the son from the West and by neighbors.

Ophthalmologic Examination.—On June 5, nine days after admission, the patient was referred to the Department of Ophthalmology for an opinion relative to the condition of the fundi. They reported, in part:

Right: Arteries somewhat contracted; some arteriovenous compression; nerve head pale and yellow in color; very heavy choroidal ring; some of the veins slightly tortuous. Foveal reflex not made out.

Left Eye: Lamina cribrosa veiled. Both the arteries and the veins somewhat contracted.

Diagnosis: Anemic fundus; angiosclerosis.

Heart Examination.—She was referred to the department of internal medicine for an examination relative to the condition of the heart. This report was made: Soft systolic at the apex—aortic roughening—possibly hemic murmur; no evidence of decompensation.

The last report was dated July 1, thirty-five days after admission. Up to that time there had been no change in the patient's general physical or neurologic condition, but the mental symptoms had become more acute, although this change was gradual, with variable intervals of relative quiet. On June 9, she insisted, and for many days repeated the idea, that she had been sent to the hospital to be a "subject," to be "ground up in a machine," and was certain that "sanitariums have to have subjects." She became restless, was frequently out of bed, refused food, saying that she could not digest it, and had periods of alternating talkativeness and uncommunicativeness. For the next two or three weeks she repeated her fears of being "ground up" almost daily, but never with a convincing air. At night, however, she occasionally paced the floor of her room and begged the nurse or the physician to put her out of the way and end her agony.

Diagnosis.—On June 25 her case was presented before a staff conference, with the diagnosis of arteriosclerotic insanity. This diagnosis was accepted by those present at the conference, and the case was entered under this classification on the hospital records. The diagnosis was reached on the basis of this summary of the case:

1. Family History: This was negative except for one feeble-minded or insane brother.

2. Gradual Onset: The onset was at least three or four years in duration.

3. Symptoms and Episodes Before Admission to the Hospital: Three or four years ago she had mild apopletic or syncopal attacks, two years ago she was sleepless, restless, lost weight, and had mental symptoms, chiefly delusions of persecution of a whimsical character and ideas of infidelity. Finally she became irritable and threatening and emotional instability was quite marked.

4. Physical Findings: There were malnutrition, pallor of the skin, flabby musculature; cardiovascular symptoms, including a faint systolic murmur at the apex. The blood pressure was systolic, 160; diastolic, 100 She had arcus senilis, anemic and angiosclerotic fundi and mild varicosities.

Laboratory Findings: These and the serologic findings were negative.

Mental Symptoms: There were emotional instability; slight disturbance of memory for recent events; circumstantial and at times, irrelevant stream of thought; delusions of persecution by her husband and of infidelity (never firmly voiced by the patient). These were the leading symptoms during the first three weeks' residence in the hospital. Gradually the mental symptoms became more severe and included terrifying delusions of bodily torture, apprehensiveness, agitation, increased psychomotor activity and transient periods of mild confusion.

Change in the Picture.—About July 1 she was again referred to the Departments of Ophthalmology and Internal Medicine. The former reported "angiosclerosis in both fundi with some appearance of anemia"; the latter reported "systolic murmur and symptoms of simple anemia, advised simple tonics." On July 3 another blood count was taken which showed the following startling results: 1,420,000 red cells; 1,530 white cells; differential count—small lymphocytes, 20 per cent.; large lymphocytes, 24 per cent.; transitionals, 9 per cent.; polymorphonuclear neutrophils, 45 per cent.; eosinophils, 2 per cent.

On the same day that this count was made, another neurologic examination revealed the following positive findings not noted in repeated previous examinations: The biceps and triceps reflexes were increased on both sides. The knee jerks and Achilles' jerks were not obtained on either side. Abdominal and epigastric reflexes were not obtained. When the tuning-fork was applied to the bony prominences, there was no perception of vibration from the patellae down, including both tibial and malleolar surfaces. Both conjunctivae were hypesthetic, the left more so than the right. The blood pressure had fallen to systolic, 102; diastolic, 60; but the pulse rate was 102. The right fundus showed what appeared to be small hemorrhagic foci. The case was again referred to the Department of Ophthalmology, which replied, on July 8:

Right Eye: Media clear. Physiologic disk plus. Lamina cribrosa veiled; nerve head oval; long axis vertical. Rings plus. Choroidal conspicuous in whole lower half of disk. Arteries somewhat contracted and pale. Some arteriovenous congestion. Veins somewhat tortuous, although no engorgement. Just below the disk, down and nasally about one third of a disk diameter, is a small round hemorrhage, petechial state, recent in part, the other portion old and has been absorbed. Along the temporal veins about the disk down, are absorbed hemorrhages, below the retina. Foveal reflex not made out.

Left Eye: Vessels essentially same as in right eye. Just one-half disk diameter below the disk along the vessels are two grayish-white spots apparently old hemorrhages. No venous hemorrhages seen in this eye. Foveal reflex not made out. Edema of the retina in both eyes.

Another blood count made four days later, July 7, showed: red cells, 1,240,000; white cells, 1,600; small lymphocytes, 20 per cent.; large lymphocytes, 30 per cent.; transitionals, 4 per cent.; polymorphonuclear neutrophils, 30 per cent.; eosinophils, 2 per cent.; normoblasts, 4 per cent.; megaloblasts, 10 per cent. There were many stippled cells and oval-shaped red cells; polychromatophilic red cells were also observed. The blood count did not change for the better, the red cells falling as low as 900,000, and the hemoglobin ranged between 30 per cent. and 50 per cent.

The mental condition became progressively worse. She became garrulous and rambling in conversation. At times she was unclear and disoriented, and became fatigued easily on the least mental effort. Terrifying delusions became more pronounced; she was to be "ground up" in terrible machines; her arms, hands and legs were to be "cut off," etc. Her mood and manner, however, were frequently out of harmony with such depressing delusions; she was cynical, playful and jested in a gruesome way. She had temporary flareups of slight improvement parallel with a slightly higher red count; but on the whole she grew steadily worse. She talked of receiving electric baths at night and of poison in her food and drink. On August 6 she became uncontrollable, had to be isolated, and died on August 19. The family refused necropsy.

Change of Diagnosis.—It is hardly necessary to state that the diagnosis was changed from that of a case of simple arteriosclerotic insanity to one of pernicious anemia with associated mental disorder. The arteriosclerotic element could not be ignored, however, and the case taken as a whole appeared, from the clinical standpoint, to be one of pernicious anemia with marked mental symptoms, particularly paranoid tendencies in a person showing arteriosclerotic symptoms.

This case shows the difficulty of differential diagnosis in pernicious anemia. The arteriosclerotic symptoms appeared first and obscured the final diagnosis. Added to this, the mental symptoms, when the case was first seen, were such as could occur in either pernicious anemia, arteriosclerosis or, as Barrett has pointed out, in other mental disorders, symptomatic of toxic conditions.

LITERATURE ON PERNICIOUS ANEMIA WITH ASSOCIATED MENTAL DISORDERS

At this point, it might be well to review briefly the literature on pernicious anemia with associated mental disorders. Until the appearance of Barrett's papers,[1] this phase of the disease had received little consideration in the literature. The mental disturbances noted in this condition had apparently been considered an incidental, unorganized terminal delirium, no dominant or characteristic psychosis having been noted.

Barrett, in his series of eleven cases, was able to determine in six of these a more or less characteristic psychopathic process, marked essentially by irritability and a definite paranoid trend, the exact nature of the specific delusions depending essentially on the particular syndrome and environment. Auditory hallucinations were observed in some cases and in others, definite confabulation. No marked deterioration was observed, the personality apparently having been relatively well preserved. Remissions in the psychic disturbance were frequently found to accompany remissions in the more essentially somatic aspect of the disease. The picture is essentially that of a gradually developing chronic condition, paralleling in a general way the progress of the actual somatic condition. Barrett suggests classifying these cases clinically among the paranoid conditions, symptomatic of toxicorganic processes.

Marcus [3] has noted that suggestive mental symptoms may manifest themselves for some time before it is possible to establish a diagnosis of pernicious anemia on the basis of the characteristic blood changes and other clinical findings. Langdon [4] directs attention to the same possibility and designates such cases prepernicious anemia. Initially, our case seems to have approximated this type.

Lurie,[2] reviewing the field in a recent article, seems to endorse Barrett's classification of the condition as a toxicorganic psychosis, remarking in addition, that the "whole delusional formation is vague, unsystematized and loosely connected." In an analysis of four cases, Lurie finds definite visual hallucinations in two cases and doubtful visual hallucinations in one other and delusions of persecution and paranoid ideas in all four.

DIFFICULTY OF DIFFERENTIAL DIAGNOSIS IN CASES LIKE THE AUTHOR'S

It was impossible to find any reference in the literature bearing directly on the question we discuss, i. e., the necessity and difficulty of differentiating between pernicious anemia with mental symptoms and arteriosclerotic insanity.

For this reason the present case is reported. During residence in the hospital, the case was looked on as one of arteriosclerosis until about a month from the day of admission, then, within the short period

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3. Marcus, H.: Psychose bei perniciöser anaemie, Neurol. Centralbl. **22**: 453, 1903.
4. Langdon, F. W.: Nervous and Mental Manifestations of Pre-Pernicious Anemia, J. A. M. A. **45**:1635 (Nov. 25) 1905.

of three or four days, the whole picture became dominantly that of pernicious anemia associated with cord symptoms and mental disorder. This called for a review or what might be called an inventory of the early history of the case as well as of the symptoms up to the time when the picture became unmistakably that of pernicious anemia. Features that had been looked on as primarily arteriosclerotic, now were just as easily interpreted as those of the so-called "prepernicious" phase of the case as a whole. These features were:

1. Tainted heredity (Barrett).

2. Gradual onset, three or four years, possibly twelve, with symptoms, especially syncope, etc., which inaccurately reported by members of the family may well have been those due to anemia as well as to arteriosclerosis. Had a blood count been taken in the earlier attacks, the interpretation of the history might have been different.

3. Delusions of a whimsical character and delusions of persecution (not necessarily characteristic of arteriosclerosis).

4. Remissions in which the patient appeared to have recovered. These symptoms running back over a period of twelve years might be only "prepernicious" symptoms. Then appeared the findings and symptoms that clouded the diagnosis; cardiovascular symptoms, including soft systolic murmur at the apex, blood pressure of 160 to 100, arcus senilis and angiosclerotic fundi.

Then occurred rapid changes certainly indicative of a case of genuine pernicious anemia: low red cell count, characteristic differential count, stippled cells, etc.; cord changes, including lost patellar reflexes and lost vibratory sense, and finally the rapid and progressive aggravation of all the mental symptoms.

In conclusion, we wish to emphasize these points:

1. Probably more cases of pernicious anemia are mistaken for arteriosclerosis than are generally recognized.

2. Patients with doubtful cases of arteriosclerosis, especially those with a history of remissions, should, as a routine procedure, have frequent blood counts made, and neurologic examinations for cord changes.

3. Careful history taking may reveal the so-called "prepernicious" symptoms in cases that otherwise might be mistaken for arteriosclerosis.

4. The lack of comment in the literature on this phase of neuropsychiatry is significant, and shows that the necessity of differentiating carefully in doubtful cases has not yet been fully recognized.

OBSERVATIONS ON THE PATH OF FARADIC SENSIBILITY *

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The present study deals with the afferent path of faradic stimuli. The evidence here presented has been largely gathered from a study of cases presenting certain neurologic lesions or psychiatric conditions, and suggests that faradic stimuli of liminal value probably follow the path of the thermal fibers, while markedly supraliminal stimuli may use other paths. As was to be expected, the postrolandic cortex is in all likelihood the arrival platform for these impulses.

METHOD USED IN MAKING TESTS

The method used eliminates subjective influences both from the patient and operator. Heretofore, quantitative sensory tests have been of only relative accuracy and most unsatisfactory. Even the ordinary qualitative sensory tests are not perfectly objective. There have been three chief objections to previous attempts at quantitating tests of sensation: First, lack of absolute accuracy, i. e., the difficulty of reproducing exactly a stimulus of given intensity at will; second, lack of objectivity, and third, the ease of deception by the patient. These three factors of error have been eliminated by employing the method devised by Martin [1] for the measurement of shocks delivered by the secondary coil of an inductorium in absolute units which he called beta units. By the use of this method any given stimulus can be reproduced exactly. The subject has no means of knowing when the stimulus is applied, as he sits in another room, and no noise warns him of the application of the stimulus. The operator has an absolute check on the accuracy of the patient's replies, by stimulating the patient with

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*Study No. 6 in a series entitled "Variations in the Sensory Threshold for Faradic Stimulation in Psychopathic Subjects."

* This work was done during the tenure of an internship at the Psychopathic Hospital, Boston.

1. Martin, E. G.: A Quantitative Study of Faradic Stimulation. I. The Variable Factors Involved, Am. J. Physiol. **22**:61, 1908. Ibid.: II. The Calibration of the Inductorium for Break Shocks, ibid. **22**:118, 1908; ibid.: III. The Measurement of Make Shocks, ibid. **24**:269, 1909; ibid.: IV. The Make and Break Key, ibid. **26**:181, 1910; ibid.: V. The Influence of Tissue Resistance and of Kathode Surface on Stimulating Effectiveness, ibid. **27**:226, 1910; ibid.: VI. The Comparison of One Inductorium with Another, ibid. **28**:49, 1911; ibid.: Measurement of Induction Shocks, New York, 1913; ibid.: On the Validity of Inductorium Calibrations, ibid. **36**:223, 1915.

shocks of varying intensity, any one of which may be repeated as often as is deemed desirable. A brief description of the method as employed by us follows.

The measurement of shocks delivered by the secondary coil of an inductorium by this method depends on the fact that with a given current passing through the primary the shock delivered by the secondary coil (on making or breaking the primary circuit) depends on the proximity of the secondary to the primary coil in any given plane. The exact relation between shock delivered and intercoil distance varies with the inductorium used. This must be calibrated for both make and break shocks.[2] Throughout this work only break shocks were used. These were delivered by the "Make-and-Break Key," [3] the advantage of which lies in the fact that it allows a uniform make or break as the case may be, and enables one to short-circuit the one or the other.

The shocks from the secondary were delivered to the patient by electrodes of the mercury-calomel-salt-solution type described by Martin, Porter and Nice [4] as the most advantageous for work with human subjects. They also found that abrasions of the fingers used in the test (or the use of platinum points thrust under the skin as electrodes) lowered the average human threshold markedly. This is a practical point of some importance in dealing with maniacal patients who often sustain slight injuries. These electrodes consist of two tubes about 5 inches long and of a diameter to admit the fingers comfortably. The wires from the secondary coil are led to the bottom of these tubes (one into each tube) over the top of the tube. The insulation of the wires is stripped for a distance of 1 or 2 cm. from their ends, which rest on the bottom of the tubes. Around the ends of these wires is a layer of mercury, over which lies a layer of calomel paste to a depth of 2 cm. The rest of the tube is filled with 15 per cent. salt solution.

The patient dips the middle and index fingers of his left hand into the electrodes and is instructed to press a telegraph key whenever he feels a shock. The operator starts with the secondary coil in such a position that it will deliver a subliminal shock, and gradually approaches it to the primary, breaking the current at short irregular intervals. Finally, the patient signals, indicating that he has felt the shock. This point and its environs are tried and retried until the point farthest from the primary is found where the patient feels practically every shock. This can be found within 2 mm. as measured on a scale which has as its zero the point where the secondary lies over the primary and encloses it. This procedure is repeated three times with ten, twenty and thirty thousand ohms, respectively, introduced into the primary circuit. From this it is evident that deception on the part of the patient is impossible. The entire procedure consumes about six minutes, on the average, though perseverance will frequently procure a reading from a subject who at first seems to offer no cooperation whatever. The resistance of the coil used in this work is 2,900 ohms. In work of the highest accuracy it is necessary to determine the resistance of the patient's skin by the somewhat tedious Kohlrausch method. This resistance has been found to be very constant, however, and in our work we felt that it was better not to determine the resistance in each case, on account of the time necessary for the procedure and the restiveness of certain patients. We have taken the skin resistance to be 2,100 ohms, the average of a large number of observations, three quarters of which fall within 300 ohms of this figure. This omission introduces only a small percentage error, especially in dealing with high threshold values. Thus, in the secondary circuit there is the resistance of the secondary coil (2,900 ohms) plus the resistance of the patient's skin (2,100 ohms) or 5,000 ohms. Our four readings of the position of the secondary coil are made with a total of five, fifteen, twenty-five and thirty-five thousand ohms' resistance in the secondary circuit. From these resistances, the value of the current passing through the primary coil, and the position of the secondary coil, the value of the threshold shock is calculated by certain formulae developed by Martin [5] as modified by Wilbur.[6] The primary current used throughout this work (except in the cases of syringomyelia) was 0.3 ampere.

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[2] Martin, E. G.: Am. J. Physiol. **22**:118, 1908; ibid. **24**:269, 1909; ibid. **28**:49, 1911; ibid. **36**:223, 1915.

[3] Martin, E. G.: Am. J. Physiol. **26**:181, 1910.

[4] Martin, E. G.; Porter, E. L., and Nice, L. B.: Sensory Threshold for Faradic Stimulation in Man, Psychol Rev. **20**:194, 1913.

Throughout this work the results have been expressed in the number of beta units required to cause sensation. One beta unit is the average amount of faradic shock necessary to cause a minimal contraction of a frog's gastrocnemius stimulated through the sciatic nerve in the classic nerve-muscle preparation.[4]

It is to be emphasized that in interpreting the results here presented, the higher the figure given (in beta units) the less sensitive is the subject.

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5. Martin, E. G.: Measurement of Shocks, New York, 1913.

6. Martin, E. G.; Bigelow, G. H., and Wilbur, G. B.: Variations in the Sensory Threshold for Faradic Stimulation in Normal Human Subjects. II. The Nocturnal Variation, Am. J. Physiol. **33**:415, 1914.

PREVIOUS WORK

Since the introduction of this method in 1908, considerable work has been done on the variations of this threshold in normal and psychopathic human subjects and in the experimental animal under various conditions. Martin, Porter and Nice[4] found that there was a practically constant threshold value for a normal human subject, tested as described above. As a result of introspective observations, they suggested that this form of sensation was allied to muscle or joint sensibility. Continuing the work on normal human subjects certain evidence was accumulated to show that this method might be used as an index of the functional condition of the central nervous system. It was found that this threshold had a definite diurnal variation[7] which corresponds closely with the curve of ergographic output as reported by Lombard,[8] and with the diurnal variation of the reaction time as recorded by Ostanikow and Gran.[9] The lack of closer correspondence with the latter curve may be ascribed to the fact that these authors made only four observations in the course of a day as compared with hourly observations in the study on this threshold. Martin, Bigelow and Wilbur[6] worked out a nocturnal rhythm which was found to agree closely with the depth of sleep as measured by Mönninghoff and Piesbergen,[10] the periods of low irritability coinciding with those of greatest sleep intensity. It was further found in this study that the second low point in irritability corresponds with the point of lowest body temperature.[11] The influence of general fatigue on this threshold is of some importance in the use of the threshold value clinically for the examination of ambulatory cases. In this study[12] it was found that men under a somewhat pressing but regular routine showed a curve of decreasing irritability as the week wore on. This curve was obtained by testing the men at the same time each day. With the intervention of Sunday and relief from their routine, the irritability increased to the point where it had begun the week before. Martin, Paul and Welles[13] found the threshold of the winking reflex in man to be about twice that of the sensory threshold and concluded that "the value of the sensory threshold depends on the state of the higher nervous centers, and that the threshold may justly be used, therefore, as an indicator of such nervous state."

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[7] Grabfield, G. P., and Martin, E. G.: Variations in the Sensory Threshold for Faradic Stimulation in Normal Human Subjects. I, The Diurnal Rhythm, Am. J. Physiol. **31**:300, 1913.

[8] Lombard, W. P.: Some of the Influences which Affect the Power of Voluntary Muscular Contractions, J. Physiol. **13**:25, 1892.

[9] Ostanikow, P., and Gran, M.: Ueber die Geschwindigskeitsveränderungen der psychischen Processe zu verschiedenen Tageszeiten, Neurol. Centralbl. **12**:290, 1893.

[10] Mönninghoff, O., and Piesbergen, F.: Messungen über die Tife des Schlates, Ztschr. f. Biol. **19**:114, 1883.

[11] Jürgenson and Liebermeister: Handbuch der Pathologie und Therapie des Fiebers, Leipzig, 1875; quoted by Pembrey in Schäfer: Textbook of Physiology, Edinburgh and London **1**: 1890. Jürgenson, T.: Die Körperwärme des Gesunden Menschen, Leipzig, 1813.

[12] Martin, E. G.; Withington, P. R., and Putnam, Jr., J. J.: Variation in the Sensory Threshold for Faradic Stimulation in Normal Human Subjects. III. The Influence of General Fatigue, Am. J. Physiol. **34**:97, 1914.

The indication that "synaptic resistance" probably has an effect on this threshold suggested certain animal experiments which showed that in the spinal cat[14] the threshold for the radial nerve-muscle preparation is almost exactly half as high as that for the flexion reflex obtained by stimulating the tibial nerve. In a further study of the variations in the threshold of the reflex arc[15] it was found that an excess of carbon dioxid raised the limen of the flexion reflex in the spinal cat to the point of its total disappearance, and that this effect varied with the percentage of oxygen present. This suggests that a study of neuropathic cyanosis may permit of correlation with this observation.

Finally, a group of experimental studies[16] on the limen of the vasomotor center showed that the intensity of the stimulus to the nervous system may make a difference in the responses to two stimuli varying in strength and not in kind. These studies also showed that the threshold of the vasomotor center is about twice that of the flexion reflex and about four times that of the nerve-muscle preparation of the spinal cat. This may possibly be taken as further evidence of the effect of the interpellation of synapses on such tests.

At this time the work was carried over to an examination of psychopathic human subjects. In a preliminary survey[17] it was found that the threshold of the psychopathic patient tends to be high, the average value in this group of cases being 223 beta units as against 100 beta units in the normal person. It was further shown that this test was a feasible one for routine use in psychopathic subjects.

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[13] Martin, E. G.; Paul, B. D., and Welles, E. S.: A Comparison of Reflex Thresholds with Sensory Thresholds. The Relation of This Comparison to the Problem of Attention, Am. J. Physiol. **26**:428, 1915.

[14] Porter, E. L.: Thresholds of Electric Stimulation in the Spinal Cat Determined by the Martin Method, Am. J. Physiol. **31**:141, 1912.

[15] Porter, E. L.: Variations in Irritability of the Reflex Arc. I. Variations Under Asphyxial Conditions, with Blood Gas Determinations, Am. J. Physiol. **31**:223, 1913.

[16] Martin, E. G., and Lacey, W. H.: Vasomotor Reflexes from Threshold Stimulation, Am. J. Physiol. **33**:212, 1914. Martin, E. G., and Stiles, P. G.: Two Types of Reflex Fall of Blood Pressure, ibid. **34**:106, 1914; ibid.: The Influence of Curare on Vasomotor Reflex Thresholds, ibid. **34**:220, 1914; ibid.: Some Characteristics of Vasomotor Reflexes, ibid. **37**:94, 1915.

[17] Grabfield, G. P.: Variations in the Sensory Threshold for Faradic Stimulation in Psychopathic Subjects: First Note, Boston M. & S. J. **161**: 883, 1914.

The larger psychiatric entities were then studied. In the manic-depressive group[18] it was found that the maniacal patients had a normal or slightly lowered threshold, and that the depressed patients showed a faradic hypesthesia which wore off as the depression disappeared. It was felt that this variation in the threshold was probably intimately connected with the fundamental cause of the variations in emotional tone in these cases. Kraepelin[19] believes that this disease is caused by a congenital predisposition to mania or depression, as the case may be, and that the alternating forms may be found either in the manic or the depressive temperament as a basis. He further believes that this predisposition is manifested in the personality of the person when he is normal. He uses the fact that maniacal attacks may be superposed on the depressive temperament as an argument for his contention that mania and depression are different degrees of the same process and not opposites as would appear. The threshold for touch, temperature and pain has been found to be high in depressed patients as measured by tests of relative accuracy. The association time in manic cases is somewhat lengthened, but is more so in depressed cases. Southard[20] has shown that there is an absence of gross brain lesions in the brains of manic-depressive subjects, and his findings and that of others have indicated the functional character of this disease. This same observer has noted lesions of the thalamus in cases[21] other than manic depressive insanity, that showed during life hyperkinesis and its psychical accompaniments. Similarly, Bevan Lewis[22] has described an absence of metabolic pigment in hyperkinetic states, and Southard and Bond[23] have shown that there is an increase of metabolic or catabolic lipoid substances in the cortex of depressed cases of manic depressive insanity. From the work on the thalamus it was suggested that the hyperkinetic states might be associated with a withdrawal of the corticothalamic "inhibitory" or "switch-setting" impulses, that is, with a "tissue simplification." It is possible from the histopathologic evidence in the opposite state that these depressions may be associated with an accumulation of catabolic products. In view of this evidence and the reactions we have demonstrated in the receptor mechanism, it was suggested as a working hypothesis that these two contrasted states in this psychosis might be brought about by a functional hypertonicity or hypotonicity (for depression and mania, respectively) of these or similar centers. This hypothesis is further supported by Sachs'[24] demonstration of the intimate relation between the thalamus and sensation. Additional suggestive evidence as to possible variations in the reactions of such centers is the demonstration that with threshold stimulation of a peripheral nerve the reflex vasomotor center[25] responds with a depressor effect, while with markedly supraliminal stimulation it responds with a pressor effect.[26]

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[18] Grabfield, G. P.: Variations in the Sensory Threshold for Faradic Stimulation in Psychopathic Subjects: II, Manic Depressive Insanity, Boston M. & S. J. **173**:148, 1915.

[19] Kraepelin, E.: Psychiatrie, Ed. 8, Leipzig, 1913.

[20] Southard, E. E.: Anatomical Findings in the Brains of Manic Depressive Subjects, Proc. Am. Psychol. Assn., May, 1914.

[21] Southard, E. E.: The Association of Various Hyperkinetic Symptoms with Lesions of the Optic Thalamus, J. Nerv. & Ment. Dis. **41**:617, 1914.

[22] Lewis, W. Bevan: A Textbook of Mental Diseases, Philadelphia, P. Blakiston's Son & Co., 1890.

[23] Southard, E. E., and Bond, E. D.: Clinical and Anatomical Analysis of Eleven Cases of Mental Disease in the Second Decade, with Special Reference to a Certain Type of Cortical Hyperpigmentation in Manic Depressive Insanity, Am J. Insan. **71**:194, 1914-1915.

In the dementia praecox group, on the contrary, no such variation with emotional tone is observable in the faradic threshold,[27] and it was concluded from this that the phenomenon of emotional tone in this group was probably governed by a different mechanism than in the manic-depressive syndrome. In the diagnosis of these cases we have largely followed Kraepelin, i. e., cases ending in deep dementia and displaying as their chief symptoms basic disorders in the personality, progressive deterioration and absence of the tendency to recover. There was found a close parallelism between cases showing symptoms of catatonia (silliness, grimacing, attitudinizing, stupor, cerea flexibilitas, negativism, etc.) and high threshold values, and between low threshold values and absence of such symptoms. These symptoms have been correlated with postrolandic lesions in these cases by Southard,[28] and this finding suggests the postcentral cortex as the "arrival-platform" of faradic sensation. Similarly, patients with somatic delusions have shown higher threshold values than patients not exhibiting such delusions. This lends support to the hypothesis that these delusions are often a result of disturbances in the receptor mechanism, although they may have their basis in fact, as shown by the lesions found at necropsy in the organs about which the delusions occurred in life.[29] Thus from this group there has been obtained evidence to show that the arrival platforms for faradic sensation are probably located in the postcentral gyrus with other forms of peripheral sensibility.

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[24] Sachs, Ernest: On the Structure and Functional Relations of the Optic Thalamus, Brain **32**:95, 1909.

[25] Porter, W. T.: The Vasotonic and the Vasoreflex Center, Am. J. Physiol. **36**:418, 1915.

[26] Porter, W. T.: The Relation of Afferent Impulses to the Vasomotor Center, Am. J. Physiol. **27**:276, 1910.

[27] Grabfield, G. P.: Variations in the Sensory Threshold for Faradic Stimulation in Psychopathic Subjects: III, The Dementia Praecox Group, Boston M. & S. J. **173**:202, 1915.

[28] Southard, E. E.: A Study of the Dementia Praecox Group in the Light of Certain Cases Showing Anomalies or Scleroses in Particular Brain Regions, Proc. Am. Medico-Psychological Assn., May, 1910; ibid.: On the Topographical Distribution of Cortex Lesions and Anomalies in Dementia Praecox with Some Account of Their Functional Significance, Am. J. Insan. **71**:382, 1914-1915.

The alcoholic group has yielded little information for present purposes, though perhaps the most important from a clinical point of view. It was found [30] that the chronic alcoholic invariably had a high threshold for this form of stimulation, and that this threshold fell after the withdrawal of the drug. In most of these cases (except those of Korsakow's psychosis and a few others) neuritis was not demonstrable by ordinary clinical methods or histologically in those that came to necropsy. It has thus been possible to demonstrate a distinct functional impairment in the receptor mechanism, by this method, without evidence of structural lesion. Apparently such a change in function can be induced by the administration of drugs to normal subjects, human or animal. E. L. Porter [31] has demonstrated increase in reflex irritability after the administration of strychnin to the experimental animal. Martin, Grace and McGuire [32] have shown a rise in this threshold in normal human subjects after the ingestion of moderate doses of acetphenetidin, and Dodge and Benedict [33] have shown a similar rise after varying doses of alcohol. The results obtained in the cases of Korsakow's syndrome showed that a severe, frank neuritis can exist without total impairment of conduction power, although these cases showed the highest values in this group, as was to be expected.

The psychoneuroses,[34] with the exception of the occupation neuroses and the traumatic neuroses, show a normal esthesia to faradism. In such cases no structural pathology has ever been demonstrated. The occupation and traumatic neuroses are, sometimes at least, associated with demonstrable lesions. However, the results in the few cases of occupation neuroses that opportunity has permitted us to examine, seems to point to a possible division of these cases into those in which there is a true neuropathy or "functional neuropathy,"[35] and those of a purely psychogenetic nature. As to the traumatic neuroses, we have only seen two cases, both of which yielded definitely pathologic values by this test. We felt that these cases probably had an organic basis, even though they were quite negative to usual clinical methods of examination. The results in this group emphasize the possibility of obtaining, by this method, evidence of true functional impairment impossible of demonstration otherwise. Clinically, the results of this test are of importance in aiding in the differentiation of these cases from the true psychoses.

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[29] Southard, E. E.: On the Somatic Sources of Somatic Delusions, J. Abnorm. Psychol. **7**:326, 1912-1913; ibid.: The Possible Correlation Between Delusions and Cortex Lesions in General Paresis, ibid. **8**:259, 1913-1914.

[30] Grabfield, G. P.: Variations in the Sensory Threshold for Faradic Stimulation in Psychopathic Subjects: IV, The Alcoholic Group, J. Nerv. & Ment. Dis. **45**:410, 1917.

[31] Porter, E. L.: Variations in Irritability of the Reflex Arc. II. Variations Under Strychnin, Am. J. Physiol. **36**:171, 1915.

[32] Martin, E. G.; Grace, C. M., and McGuire, J. H.: The Influence of Drugs on the Human Sensory Threshold, J. Pharmacol. & Exper. Therap. **6**:257, 1915.

[33] Dodge, R., and Benedict, F. G.: Psychological Effects of Alcohol, Publication No. 232, Carnegie Institution of Washington, Washington, D. C., 1915.

[34] Grabfield, G. P.: Variations in the Sensory Threshold for Faradic Stimulation in Psychopathic Subjects: V, The Group of Psychoneuroses, J. Abnorm. Psychol. **11**:328, 1916-1917.

The epileptics, senile and syphilitic cases have not been considered, and will be taken up later together with the data derived from other cases showing definite neurologic lesions.

DATA

Data for the present study were obtained from 612 observations on 261 cases chosen at random from the admissions to the Psychopathic Hospital, Boston. Patients obviously unsuitable were not attempted.

In this group of cases (see Table 1), as in the first group studied, it is seen that the threshold of the psychopathic patient tends to be high, the value in the entire series averaging 197 beta units. This tendency of the psychopathic patient to have a faradic hypesthesia is not due to his mental incapacity as such. The only results here reported are those in which there was no doubt as to the validity of the reading, i. e., those in which conscious or unconscious deception on the part of the patient tested could be absolutely ruled out.

The Peripheral Neuron.—Cases such as those showing frank neuritides invariably yielded high threshold values. This is true whether there was in addition to the peripheral lesion a central lesion or not. As an example of the former are the cases presenting Korsakow's syndrome, and of the latter, cases of alcoholic neuritis without demonstrable central lesions.

As has been pointed out above, in many cases this method shows an hypesthesia in cases where such an impairment has not before been suspected. In the four cases of acroparesthesia studied (these are classified with the neurologic group in Table 1), we found uniformly high threshold values, confirming the suspicion from previous studies that the peripheral receptor system was impaired in this disease. Oppenheim[36] considers that this may possibly be a spinal cord condition, but that "it is probably due to a condition of irritation in the vasomotor centers, by which the arteries are contracted and the nutrition of the sensory nerve endings in the extremities are impaired. The same author also describes these cases as being negative to sensory tests or showing "merely slight decrease of the sensibility of the finger tips."

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[35] Southard, E. E., and Solomon, H. C.: Occupation Neuroses, in Handbook for Occupational Diseases, Ed. by Hanson, Washington, D. C.

TABLE 1.—Data Available in 261 Cases Studied *

Diagnosis	Number Studied	Number of Observations	Limits of Variation in Beta Units	Average
Delirium tremens	22	32	85– 493	241
Acute alcoholic hallucinosis	16	25	46– 545	269
Korsakow's psychosis	6	18	156–1560	443
Unclassified alcoholic psychoses	14	26	86– 600	249
Dementia praecox	51	87	61– 777	186
Manic depressive insanity	25	66	23–1661	237
Psychoneuroses	30	113	41– 785	179
Epilepsy	8	12	108– 408	217
Senile group	7	11	124– 738	274
Defective group	12	22	65– 364	179
General paresis	18	50	41– 589	271
Huntington's chorea	2	4	141– 650	307
Neurologic	20	104	38–1881	292
Disturbances of the internal secretions	4	6	115– 234	147
Unclassified	26	42	66– 621	210
Total	261	612	23–1881	197

* The averages in the table are without any corrections for wrong diagnoses or complicating conditions, such as alcoholism, which would tend to raise or lower the threshold, thus throwing such a case out of alignment with its group.

From the study of this group we may safely conclude that lesions of the peripheral neuron cause a faradic hypesthesia, though we have not yet encountered clinically a faradic anesthesia from such a lesion, no matter how severe.

The Spinal Neuron. — Cases showing lesions of the posterior columns have for the most part high threshold values. We have examined five cases of this type: two of tabes dorsalis, two of taboparesis and one of ataxic paraplegia. Case No. 4656[37] showed clinically an hypesthesia over the ulnar distribution of both hands and a hyperalgesia over both arms. The grip of the right hand was stronger than that of the left. Using the little and ring fingers the threshold value on the left was 326 and on the right 574 beta units; using the index and middle fingers (these were the ones used throughout this work unless otherwise stated) the threshold on the left was 159, and on the right 383 beta units. Thus it is seen that in this case the diminution in sensory acuity on the ulnar side is accompanied by a diminution in faradic sensibility. The values are higher on the side of the body showing the greater muscular power in this case. Case No. 3931, diagnosed as taboparesis, showed diminution of sensibility to pain over the legs and chest, but no sensory changes over the arms. This case showed a threshold of 433 beta units. However, the cortical involvement in this case must be borne in mind. The case of ataxic paraplegia studied showed a threshold of 178 on the right and 143 on the left. Cutaneous sensation was clinically unimpaired. The reflexes were greater on the left than on the right, but it was noted that this is a doubtful finding. The case seems to make negative the effect of posterior column sclerosis, per se, in raising this threshold. The lesions in the spinal ganglion in tabes may well be the chief cause of the raised threshold in these cases, assuming that such lesions most probably affect more than the fibers of deep sensibility.

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[36] Oppenheim, H.: A Textbook of Nervous Diseases, Ed. 5, 1911. Trans. by Alexander Bruce, New York, 1911.

[37] Case numbers refer to the record numbers in the Psychopathic Hospital, Boston.

TABLE 2.—Figures in Beta Units Obtained from Examination of All the Fingers in Pairs of a Case Showing Residua from an Attack of Botulism

Fingers Used	Right	Left
Thumb and index	282	127
Index and middle	293	128
Middle and ring	252	163
Ring and little	255	217
Little and thumb	361	214
Average	289	170

Further evidence as to the spinal path is obtained from the study of a case showing certain residua following an attack of botulism. The case has been reported [38] elsewhere. At present this patient has a hemithermo-anesthesia on the right including the entire body up to the ramus of the jaw, above which sensation is normal. On this side there is also a slight hyperalgesia and a slight hyperhidrosis. Extremes of heat and cold are felt as pain. On the left the threshold of all the fingers taken in pairs (see Table 2) averaged 170 beta units and showed an increasing hypesthesia as the ulnar side was approached. The right hand, on the contrary, similarly tested averaged 289 beta units, the lowest value obtained on this hand being 35 beta units above the highest on the left. This patient was not only not psychopathic but was trained in introspective observation. Subjectively he made the interesting observation that on the left the sensation was localized in the ends of the fingers, whereas on the right it was located between the two fingers tested and felt more like a muscle twitch.

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[38] Stiles, P. G.: A Case of Apparent Food-Poisoning of the Type Known as Botulism or Allantiasis, J. A. M. A. **41**:2301 (Dec. 27) 1913.

Cases of Syringomyelia.—The results of two cases of syringomyelia tested in various ways properly fall in this section.

Case 4166.—P. W., aged 40, had no mental symptoms except irritability of temperament. Blood pressure was: systolic 115, diastolic 75. The Wassermann tests of blood and spinal fluid were negative. The pupils showed a slight horizontal nystagmus, especially the left. They reacted normally to light and accommodation. Biceps and triceps reflexes were absent. The abdominal reflex was absent. The Babinski and Oppenheim signs and ankle clonus were present on both sides. There was patellar clonus on the right. Knee jerks and ankle jerks were equal and exaggerated on both sides. The cremasteric reflex was absent. There was slight swaying in Romberg's position. Tactile sensation (light touch) was everywhere unimpaired. Temperature and pain sensation were lost everywhere except over the face. Ordinary clinical laboratory tests were essentially negative.

Case 3729.—K. M., aged 54. The only mental symptoms that this patient showed were some persecutory delusions. Blood pressure was: systolic 170, diastolic 110. The Wassermann tests of the blood and spinal fluid were negative. There was marked tremor of the extended hands; marked scoliosis with convexity to the right—a true rotolateral curvature. Pupillary reactions were normal. The conjunctival reflex was absent. Biceps and triceps reflexes were absent. The abdominal reflex was absent. The knee jerks and ankle jerks were hyperactive but equal. Babinski's sign was absent. Oppenheim's sign was inconstant. There was ankle clonus on both sides, better sustained on the right. Romberg's sign was absent. Tactile sensation (light touch with cotton) was everywhere unimpaired. On the right side, however, she was unable to distinguish the head from the point of a pin. There was temperature anesthesia over the right arm and hand, and an apparent slight disturbance everywhere on both sides. Pain sense was absent on the right side and somewhat disturbed on the left. Deep sensation was well preserved everywhere. Muscle sense was poor. There was slight dysdiadokokinesia on the right. Ordinary clinical laboratory tests were essentially negative.

A number of observations (see Table 3) on both these cases show that Case 4166 had an average threshold on the right of 396 beta units as compared with 505 beta units on the left. Case 3729 averaged 928 beta units on the right and 472 on the left; the latter showed comparable figures in one observation made on the feet. It is interesting to note that both of these patients could distinguish painful faradic stimuli from threshold stimulation. These were roughly estimated on Case 3729 in the feet and were found to be 875 beta units on the left and 4,000 on the right. As a comparison, it is to be noted that the strength of stimulus at the threshold for these cases is, when applied to a normal person, distinctly painful.

Cases of Cortical Lesions.—In this section we will consider those patients whose lesions are practically wholly cortical with evidence of a definite preponderance of damage on the one side or the other. In the cases of general paresis (see Table 4) there was no sensory impairment demonstrable by the usual clinical methods. It will be seen from the table that the more affected side averages 333 beta units as compared with 175 on the less affected side. In every case the threshold of the more affected side is higher than on the less affected side. Case P. B. has the following history:

TABLE 3.—Observations on Two Cases of Syringomyelia. No. 3729 Had Clinical Evidence of Advancement of the Process in the Period of Observation

Case Number	Parts Tested	Right	Left	Date
3729	Index and middle fingers	845	517	10/28/14
3729	Index and middle fingers	513	131	11/ 2/14
3729	Index and middle fingers	766	265	11/ 9/14
3729	Index and middle fingers	794	333	11/17/14
3729	Index and middle fingers	345	195	11/30/14
3279	Index and middle fingers	...	246	12/ 6/14
3729	Index and middle fingers	1590	585	12/10/14
3279	Index and middle fingers	692	679	1/ 2/15
3729	Index and middle fingers	1881	1302	1/ 3/15
3729	Feet	989	585	12/10/14
	Average	928	472	
4166	Index and middle fingers	302	560	1/11/15
4166	Index and middle fingers	462	829	1/12/15
4166	Index and middle fingers	418	307	1/14/15
4166	Index and middle fingers	379	335	1/16/15
4166	Index and middle fingers	418	444	1/17/15
	Average	396	505	

TABLE 4.—Cases of Cortical Lesions

Case Number	Diagnosis	Threshold Right	In Betas Left	Side More Affected
3920	Imbecile	119 105	172 120	Left
3699	Cerebrospinal syphilis	270	189	Right
3822	General paresis	271	196	Right
3832	General paresis	182	255	Left
3674	General paresis	370	260	Right
3857	General paresis	275	215	Right
P. B.	Cerebral thrombosis	400 843	150 158	Right

He was a man of 50, who in the early part of 1912 developed a spastic paralysis of the right side. A tumor in the left motor area was suspected. He was operated on and at operation a large mass of obviously extraneous tissue was removed from the left cortex. The process, it was seen, had also involved the postcentral gyrus. On pathologic examination it was found that this mass was composed of scar tissue, and it was decided that the process was in all probability an extensive cerebral thrombosis, apparently quite sharply localized to the left side. He made an excellent operative recovery, and went home in July, 1912. In December, 1914, he noticed a slight increase in the symptoms on the right. The left side gave him no trouble throughout the period that he was under observation.

This patient, when first tested in 1912, showed a threshold of 150 beta units in the left hand and 400 in the right. In 1914, on his return, he showed a threshold of 158 beta units on the left and 843 on the right. In addition to the above, a number of patients have been tested who showed diffuse cortical lesions. These showed invariably a high threshold value and include the senile group, huntington's chorea, various syphilitic lesions including other cases of general paresis than those mentioned above (see Table 1). These cases will be discussed later.

DISCUSSION

It was to be expected that it could be shown that faradic stimuli used one of the paths described by Head, Rivers and Sherren.[39] An absolutely dogmatic statement as to the path of these impulses is not warranted by the evidence now at hand, and further work should be done to firmly establish such a path.

Several hypotheses suggest themselves. Faradic stimuli may use all paths equally and therefore the impairment of any sensory path may raise the threshold value to this form of stimulation. However, there are serious objections to this in that the hypesthesia caused by lesions of pain and temperature fibers far exceeds that in which only other systems are involved. This suggests the hypothesis that such fibers are the path normally used by these stimuli. However, the finding of some sensory impairment in many cases considered chiefly motor would suggest the possibility of the path associated with the motor nerves, i. e., the fibers conveying deep sensibility. Such an hypothesis is supported by the findings in cases of peripheral neuritis showing almost wholly motor symptoms, such as contractures, paralyses, etc. Another implication is found in the cases showing posterior column sclerosis which invariably showed an hypesthesia.

Our feeling is that the degree of hypesthesia associated with lesions affecting the thermal fibers, as shown in the cases with dissociation of sensation, is so marked as compared with that caused by apparently equal destruction of other sets of fibers that we are justified in concluding that faradic sensibility is carried largely, if not wholly, by these fibers in the lateral columns.[40] The hypesthesia found associated with other spinal lesions can be explained by the statement that these fibers may be affected without clinical evidence and that the delicacy of this reaction has discovered hitherto unsuspected involvement. This is not too great an assumption in view of the fact that impairment of sensation has been demonstrated by this method in patients thought to have normal sensibility. We cannot exclude the other fibers as a path in stimuli of great intensity, as there may be a spread or a change in reaction — each of which possibilities has analogues in the behavior of the nervous system. We have not mentioned the possibility of a separate set of fibers carrying this sensation as the teleological argument against such fibers is practically conclusive. We believe, therefore, that the evidence points strongly to the thermal fibers as the path of this form of stimulation.

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[39] Head, H.; Rivers, W. H. R., and Sherren, J.: The Afferent Nervous System from a New Aspect, Brain, **28**:99, 1905. Head, H., and Sherren, J.: The Consequences of Injury to the Peripheral Nerves in Man, Ibid., p. 16. Head, H., and Holmes, G.: Sensory Disturbances from Cerebral Lesions, Ibid., **34**:102, 1911-1912. Rivers, W. H. R. ,and Head, H.: A Human Experiment in Nerve Division, Ibid. **31**:323, 1908.

[40] Head, H., and Thompson, T.: The Grouping of Afferent Impulses in the Spinal Cord, Brain **29**:537, 1906. May, W. Page: The Afferent Path, Ibid., p. 742.

Presumably, then, we should find evidence that the postcentral convolution is the arrival platform for the impulses that we are studying. The evidence on this point, while mostly indirect, is, we believe, conclusive. That this gyrus contains the sensory projection areas was shown experimentally by Cushing[41] (see also Bergmark [42]). We have derived evidence on this point from a study of a large variety of cases showing various cortical pathology, some diffuse and some localized. Those cases showing widespread general cortical pathology invariably showed high threshold values.

The cases of epilepsy studied (see Table 1) showed an average threshold of 217 beta units — a marked disturbance of the receptor mechanism for this form of stimulation which cannot be accounted for by peripheral lesions. Unfortunately, the evidence of cortical pathology is not conclusive. Southard has studied the glia tissue in epilepsy,[43] and he suggests that the epileptic discharge really depends on the simplification of the course of the sensorimotor afferent impulses by a reversion of this synapse to the spinal type. This may be correlated with our results in so far as they indicate the possibility of increased synaptic resistance between afferent impulses and the paths leading to the nonmotor areas of the cortex, and thus imply the possibility of lowered synaptic resistance to impulses from the periphery to the motor cortex. The few normal values obtained in this group may be correlated with the possible true idiopathic epilepsy.[44] However, while most of the research up to the present has been unsatisfactory,[45] the cause of this condition is likely to be found in the abnormal excitability of the efferent path. If this proves to be true, an efficient reaction of the organism would be a decreased excitability of the afferent path or an increase in the resistance of the synapses between the two in the cortex. It seems not unlikely that the faradic hypesthesia here demonstrated represents the effects of some such protective reaction.

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[41] Cushing, H.: A Note on the Faradic Stimulation of the Post cerebral Gyrus in Conscious Patients, Brain **32**:44, 1909.

[42] Bergmark, G.: Cerebral Monoplegia with Special Reference to Sensation and to Spastic Phenomena, Brain **32**:342, 1909.

[43] Southard, E. E.: On the Mechanism of Gliosis in Acquired Epilepsy, Am. J. Insan. **64**:607, 1908.

[44] Thom, D. A., and Southard, E. E.: An Anatomical Search for Idiopathic Epilepsy, Rev. Neurol. & Psychiat. **13**:471, 1915.

In the senile group of psychoses high values have been found. These cases are usually associated with gross brain lesions at necropsy. In previous work, however, we have gained the impression that there is a decreasing sensitivity with increasing age (though not to what we have considered pathologic values). Such an hypesthesia as this may be accounted for by impaired nutrition of nervous tissue either in the periphery or centrally either due to frank arteriosclerosis or to the "narrowing of the capillary field" which Mackenzie [46] has pointed out.

Another small group of cases in which there is generalized cerebral involvement is that of Huntington's chorea. Oppenheim [36] states that there are "disseminated, miliary, encephalitic foci especially in the cortex and the motor region," and Spiller [47] noted a proliferation of the neuroglia, especially in the second and third layers of the cortex chiefly in the motor area. He also conveys the impression that this is a diffuse process. These cases showed a constantly high threshold, and with such cerebral findings at necropsy the possible relation of the effect of lesions of what are probably associational regions on sensation, seems worth further study.

In respect to general cerebral conditions, we have had the opportunity of examining two cases in which the clinical signs were only of increased intracranial pressure. Both these cases showed a high threshold value on both sides without preponderance of hypesthesia on the one side or other. The one proved to have a left cerebellar tumor and the other a large glioma occupying chiefly the midbrain.

From the foregoing paragraphs it is seen that general lesions of the cortex and various changes of the relations inside the cranium apparently cause an hypesthesia. The cases mentioned which showed a preponderance of lesion on one side or the other and a corresponding preponderance of hypesthesia complete the evidence that the postcentral cortex is the arrival platform for these impulses. In addition, there is evidence concerning this in the study of the dementia praecox group which brings out a striking parallelism between the postrolandic lesions discovered in catatonic cases by systematic brain photography[48] and high threshold values.

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[45] Vogt, H.: Epilepsie, in Handbuch der Psychiatrie, Spezieller Teil 1ste Abteilung, Ed. by G. Aschaffenberg, 1915.

[46] Mackenzie, James: Diseases of the Heart, Ed. 3, London, 1914.

[47] Spiller, W. G.: Symptomatology, Pathology and Treatment of Choreiform Movements, J. A. M. A. **44**:433, 1905.

We have, then, evidence that threshold faradic sensation is carried by the peripheral neuron following the path of the thermal fibers in the cord and arriving at the cortex in the postcentral gyrus. Lesions of any portion of this path produce an hypesthesia to faradic stimulation.

In conclusion, we wish to emphasize the possibilities of further study of the nervous system by this method, both experimentally and clinically, as a means of throwing some light on the variations in the reactions of the nervous system under various physiologic and pathologic conditions, the latter whether structural or functional.

SUMMARY AND CONCLUSIONS

1. The method of Martin for determining the strength of shocks delivered by the secondary coil of an inductorium in absolute units is described as used by us in the determination of the sensory threshold for faradic stimulation in 261 cases (612 observations). The advantages of this method over previous methods is pointed out.

2. Previous work with this method is reviewed.

3. Clinical considerations have been purposely omitted from this discussion, but it is to be noted that the epileptic, senile and syphilitic groups of cases tend to have a faradic hypesthesia. These groups have not been reported elsewhere.

4. Cases of acroparesthesia and Huntington's chorea, though few in number, showed constantly a pathologically elevated sensory threshold value in beta units, i. e., the number of units of shock required to cause sensation.

5. Certain suggestions as to the mechanism of the relation of the variation of sensibility with that of emotional tone in the cases of manic-depressive insanity have been made.

6. From a study of this pathologic material, the hypothesis is suggested that faradic stimuli use the path of the thermal fibers in the cord and presumably in the peripheral nerves, arriving in the cortex at the postcentral gyrus. Lesions affecting any part of this path either

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[48] Southard, E. E.: On the Direction of Research as to Analysis of Cortical Stigmata and Focal Lesions in Certain Psychoses, Trans. Assn. Am. Physicians **29**: 1914.

functionally or structurally cause an hypesthesia to this form of stimulation. It was impossible to determine whether such stimulation used the thermal receptors in the skin.

It is a pleasure to acknowledge my indebtedness to Dr. E. G. Martin for his help and to Drs. H. A. Christian, H. Cushing, W. E. Paul, and E. W. Taylor for the privilege of examining certain cases reported here.

In conclusion I wish to express my sincerest thanks to the late Dr. E. E. Southard, not only for placing the clinical material at my disposal, but also for his kindly interest and invaluable and stimulating suggestions throughout this work.

University Hospital.

A SIMPLE METHOD FOR RECORDING CHANGES IN CUTANEOUS SENSIBILITY *

LAURENT FEINIER, M.D.
NEW YORK

The New York Neurological Institute and several other New York hospitals have adopted a method for the recording of changes in cutaneous sensibility, which on account of its simplicity is, we believe, worthy of being used as a standard. The method was suggested by Dr. Elsberg, and has served in our institution to obviate the difficulties often encountered in the understanding of the records of sensory disturbances. Whenever such a chart is examined, whether it be at the bedside or in a medical journal, it often requires considerable study before the meaning of the markings by which the different disturbances of sensibility are characterized are understood.

The sensory sheets we use at the New York Neurological Institute are of a uniform size to fit the hospital bedside chart, i. e., 8⅝16 inches wide by 10 11/16 inches long. The chart has printed on it a human figure in the anterior and posterior position (see chart). The charts are printed on paper of three colors—the white for the tactile sensation, the red for temperature and the yellow for pain. In the upper right hand corner of the sensory sheet of appropriate color is printed the sensation to be recorded. Below the body outlines is printed a key to the method of making sensory markings so that a physician who is not accustomed to making the records according to the standard can easily understand how to record the sensory disturbances.

For total loss of sensation transverse lines are made and, as transverse lines mean lost sensation, it is of litttle consequence how near together or how far apart the lines are drawn. It has, however, been found advantageous to make the lines ⅛ to ¼ inch apart to facilitate reading. Thus, transverse lines drawn on the figures of the white sensation sheet mean anesthesia; transverse lines drawn on the figures of the red sheet (temperature) mean thermal anesthesia; transverse lines drawn on the figures of the yellow sheet (pain) mean analgesia.

Diminished sensation is represented by three varieties of oblique lines running from the right downward and to the left; for slight diminution these oblique lines are spaced ¼ inch apart; for moderate diminution ⅛ inch apart, and for marked diminution 1/16 inch apart. It is not to be hoped that mathematical preciseness in spacing the lines exactly according to the above measurements will always be maintained,

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* From the Clinics of the Neurological Institute, New York.

but if they are approximated a quick survey should convey an intelligent grasp of the sensory situation. Thus, oblique lines mean diminished sensation, and the nearer together the lines are the more marked is the diminution in sensation. As it is often advisable to record separately disturbances in the recognition of heat and cold, oblique lines running from right to left and downward are used to denote disturbances in the recognition of heat, and those running from left to right and downward are used to denote disturbances in the recognition of cold.

In a similar way, vertical lines show hypersensitiveness to touch, pain or temperature. The more closely together the vertical lines are, the more marked is the hypersensitiveness; thus, the vertical lines on the white sensation chart record the presence and degree of hyperesthesia; vertical lines on the yellow chart record the various degrees of hyperalgesia and those on the red chart hypersensitiveness to hot and cold.

The accompanying three charts, on which are recorded the sensory disturbances of the integument exhibited by a patient with a spinal cord tumor in the upper dorsal region, will serve as an illustration of the method used to record sensory changes.

On the white or tactile chart, it will be noted, is an anesthesia of the lower extremities extending almost to the umbilicus anteriorly and to the level of the first lumbar vertebra posteriorly. Anteriorly, above the lost sensation, there is over the upper abdomen and lower thorax an area of slight diminution in tactile sensation, and just below the left nipple an area of marked diminution. Posteriorly, sensation is lost completely to the seventh dorsal vertebra on the right with an area of moderate diminution from the level of the sixth dorsal vertebra to the first lumbar on the left.

On the yellow or pain sheet, there is an analgesia of the lower extremities with an area or band of slight hyperalgesia posteriorly at the level of the angles of the scapulae, and between this and the analgesia on the right posteriorly an area of slight hypalgesia.

Anteriorly similar changes are noted, and there is in addition a band of moderate and slight diminution in pain sense extending from the left to the right, and above this a band of moderate hyperalgesia.

Similarly, on the red or thermal sense chart there is recorded a total loss of sensation in the lower extremities, above this an area of moderate diminution, and extending around the figure above the areas of lost and diminished thermal sense, there is a band of moderately hypersensitive thermal sense.

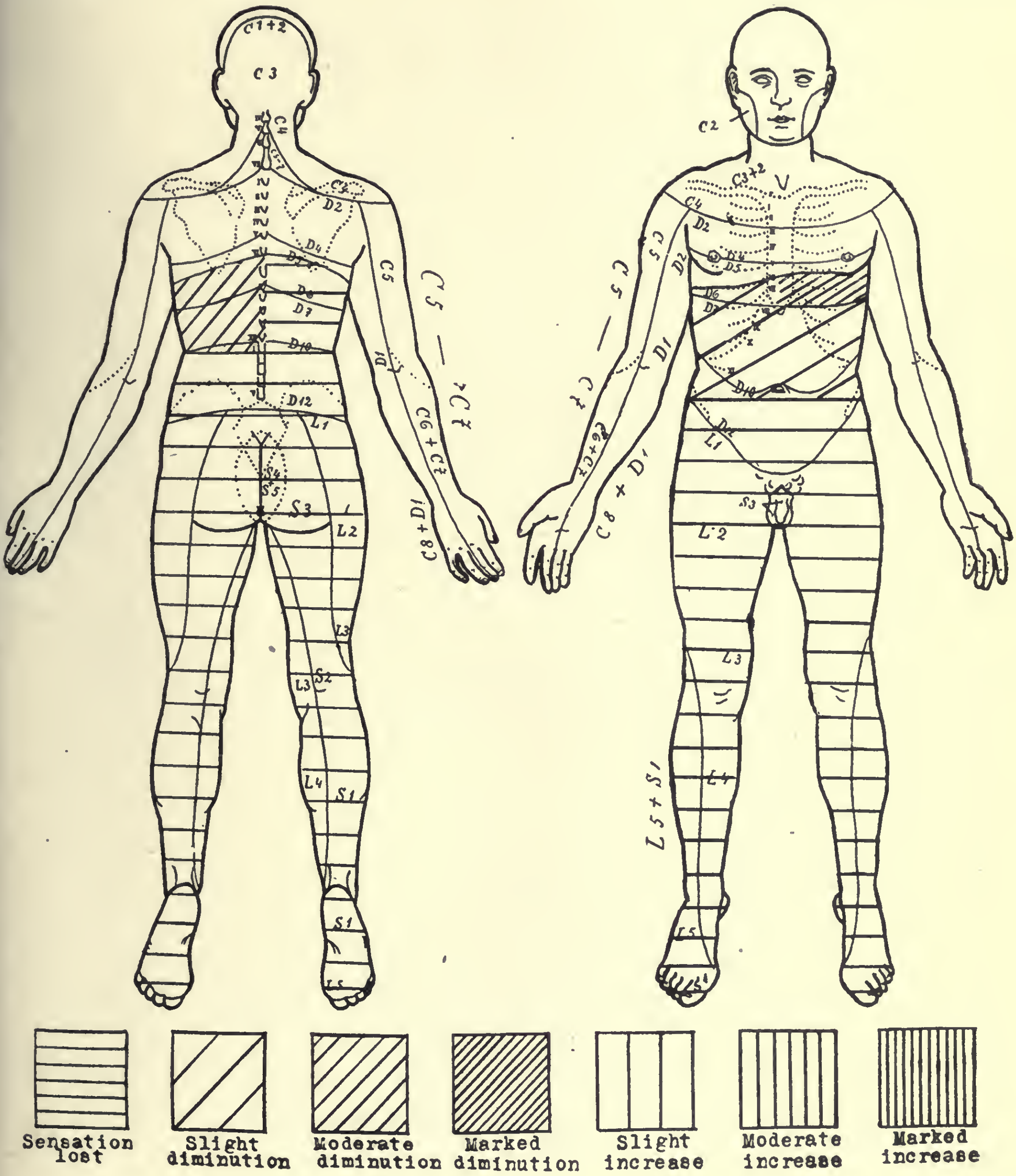
149 East Sixty-Seventh Street.

TACTILE

THE NEUROLOGICAL INSTITUTE, New York

Name: Mr. A.

Date: Oct. 18, 1919.

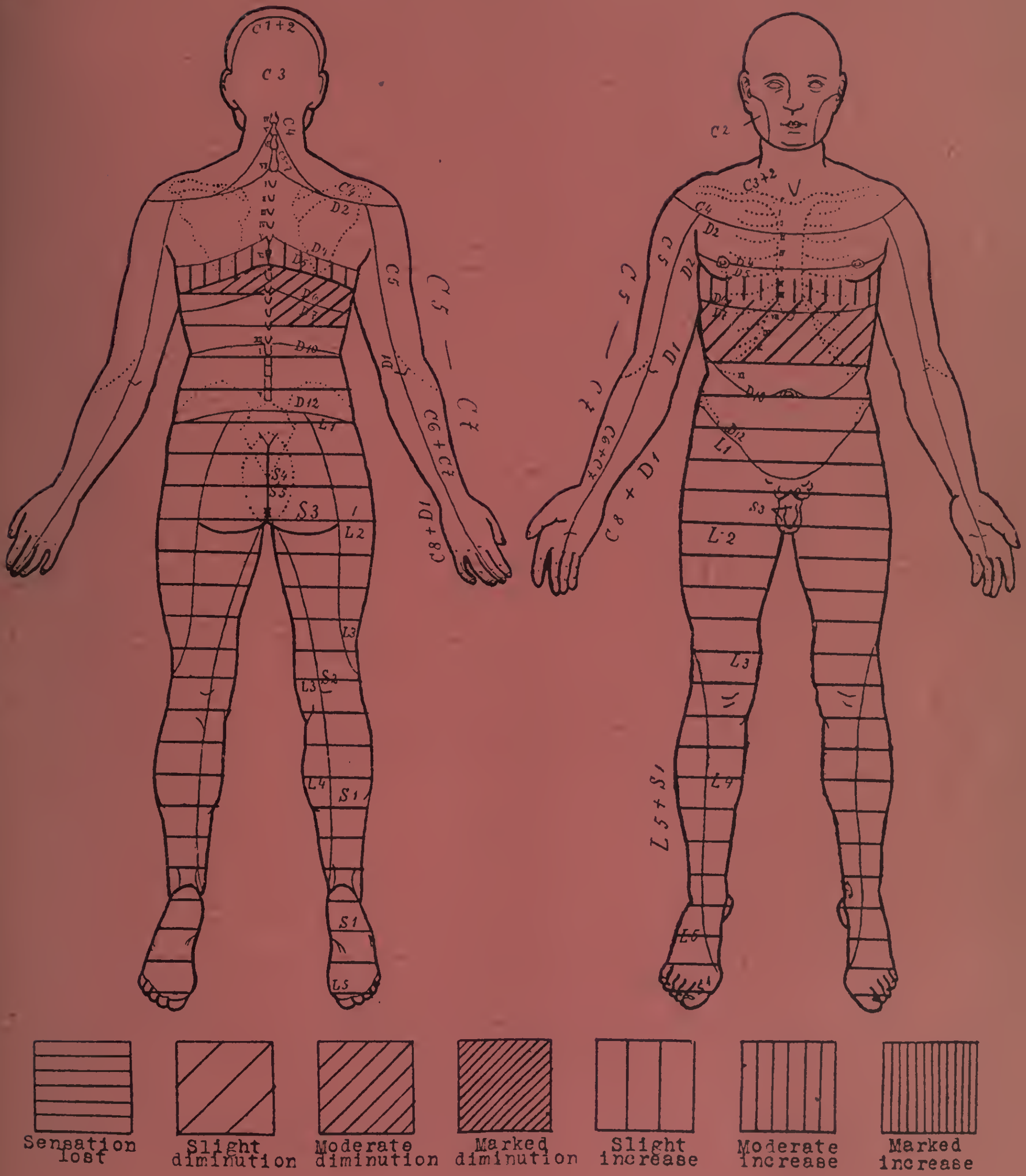


TEMPERATURE

THE NEUROLOGICAL INSTITUTE, New York

Name: Mr. A.

Date: Oct. 18, 1919

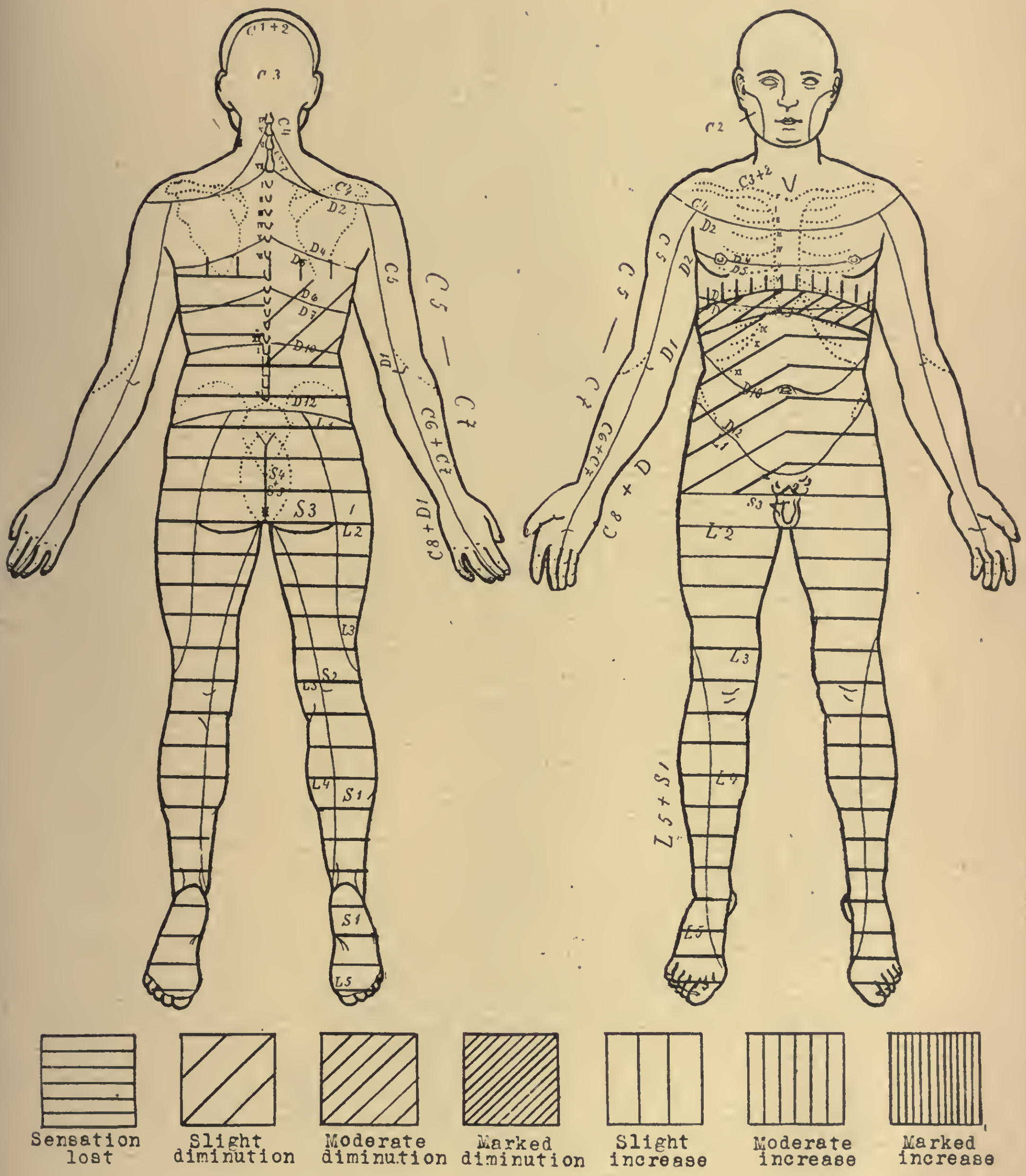


PAIN

THE NEUROLOGICAL INSTITUTE, New York

Name: Mr. A.

Date: Oct. 18, 1919.



News and Comment

AMERICAN NEUROLOGICAL ASSOCIATION ELECTS OFFICERS

At the last meeting of the American Neurological Association, June 1, 2 and 3, the following officers were elected: president, Dr. Sidney I. Schwab, St. Louis; first vice president, Dr. Hermon C. Gordinier, Troy, N. Y.; second vice president, Dr. Henry H. Donaldson, Philadelphia; secretary-treasurer, Dr. Frederick Tilney, New York; councilors, Drs. J. Ramsay Hunt, New York and Henry M. Thomas, Baltimore; councilors holding over, Drs. E. W. Taylor, Boston, D. J. McCarthy, Philadelphia, and T. H. Weisenburg, Philadelphia. The time and place of the next meeting will be determined by the council about next February.

ASSOCIATION FOR RESEARCH IN NERVOUS AND MENTAL DISEASES

A new association under the above title has been organized through the initiative of the New York Neurological Society.

Its membership was originally to have consisted of all members in good standing of the neurological societies of the cities in fairly close proximity to New York, namely, Baltimore, Boston, Brooklyn, Philadelphia and Washington. However, invitations have since been extended to the neurological societies of the cities beyond these limits, and preliminary favorable responses have been received from Chicago, Pittsburgh and San Francisco.

The Organization Committee is composed of the following: Drs. Pearce Bailey, Charles L. Dana, J. Ramsay Hunt, Smith Ely Jelliffe, George H. Kirby, Foster Kennedy, Bernard Sachs, Israel Strauss, Frederick Tilney, Walter Timme, Lewellys F. Barker, E. W. Taylor and T. H. Weisenburg.

The aim of the organization is to foster research in neuropsychiatric diseases. Its meetings will be held yearly and are to be conducted by a commission whose members will act as an investigating body for the presentations and theses submitted at each annual meeting. The subject for each annual meeting will be announced from one to three years in advance in order to enable the necessary laboratory and other research work to be conducted.

At each annual meeting there will be but one subject for discussion, all phases of which will be covered and all discussion and all activity at the meeting will be directed toward the solution of the problems presented. At the conclusion of the annual meeting the commission will prepare and publish such data and views presented at the meeting as they deem fit, with their own interpretation of the various papers presented. Such publication is to be of broad and comprehensive scope.

The Commission has decided on New York for the first meeting, during the Christmas week of 1920. The subject for discussion is "The Acute Nonsuppurative Infections of the Nervous System." Already a more or less complete program has been decided on by the Program Committee, of which Dr. Charles L. Dana is Chairman.

The financial burdens will be borne by the members. Each member is to pay a fee of $5 yearly to the Secretary-Treasurer of his local neurological society, who will transmit all these fees by October of each year to the Secretary-Treasurer of the Research Association, Dr. Foster Kennedy. This fee will entitle the member to be present at the annual meetings and probably also to receive a copy of the transactions when these are completed.

The organization of the Research Association is practically completed and the details will be presented at the business session of the annual meeting next December. Dr. Walter Timme is chairman of the Organization Committee and Dr. Frederick Tilney is chairman of the Committee of Arrangements for the first annual meeting.

Abstracts from Current Literature

ZUR PATHOLOGIE DER EPILEPSIE NACH SCHADELSCHUSSVERLETZUNGEN (PATHOLOGY OF EPILEPSY FOLLOWING GUNSHOT WOUNDS OF SKULL). E. Redlich, Ztschr. f. d. ges. Neurol. u. Psychiat. **1**:48, 1919.

Redlich reports fifty-seven cases of epilepsy following head injuries of warfare. In eighteen of these the attacks were designated as early attacks, occurring at the time of injury or soon following operation; of the remaining thirty-nine cases in which late attacks occurred, twenty-six developed in the first six months (66.7 per cent.), and eight cases developed in the second six months (20.6 per cent.), making a total of 87.3 per cent. within the first year. The frequency of the onset of the late attacks during the first six months has been emphasized by Voss and Jolly. A survey of the literature of traumatic epilepsies of warfare is made with especial reference to a statistical study of frequency of occurrence.

Epilepsy occurring after war injuries is characterized by relatively infrequent attacks; the author has not observed a status epilepticus. Among his series he reports one case of a single attack occurring early after trauma; in three others, single late attacks, occurring fifteen, eighteen and twenty-six months later, respectively.

The disposition for epilepsy is the head injury. More frequent than in ordinary epilepsy, the exciting factors are evident. Most important is the direct irritation of the motor cortex, producing typical jacksonian epilepsy. These attacks frequently cease after surgical removal of the irritating factors, bone fragments, bullet particles, hemorrhage, etc., but they may continue or recommence after an interval of time. Attacks may occur during or following surgical intervention, not directly involving the motor cortex (Böttiger). Redlich reports a considerable number of cases in which the attacks followed early operation. He interprets these attacks as due to mechanical irritation of the motor zones, due to the trauma of operation, hemorrhage in the subarachnoid space and in the cortex, etc.

Although early operation, débridement, removal of fragments, have been generally recommended to prevent future attacks, in the experience of the writer this procedure cannot be considered as offering certain protection, because splinters, and especially metal splinters, cannot be entirely removed by surgical procedure, even by a secondary operation. The covering in of a bone defect following skull injury is frequently followed by epileptic attacks. This procedure has been suggested as preventing such attacks. Other exciting causes are: inflammatory reaction of the wound, sensory stimuli to parts involved in the epileptic contractions, such as application of the faradic current in recent hemiplegias, massage, passive motion and undue amount of exercise; local application of heat over the traumatic area and exposure to the sun's rays. Absolute abstinence from alcohol should be the rule in these cases.

Most often the epileptic attacks are of the jacksonian type, although all forms are observed, such as the form beginning with jacksonian epilepsy and becoming generalized with loss of consciousness. Attacks of vertigo, with sensations of heat over the body, nausea and transient loss of consciousness, is a symptomatology frequently met with. Psychic equivalents were not observed, and postparoxysmal psychic disturbances were comparatively rare. Aurae of motor and sensory origin were frequent, and would often give timely and useful warning for protection or even abortion of the attack. In the jacksonian type it appears that there is a predisposition for the onset of the spasm in certain anatomic regions; in the author's cases the onset was much more frequent in the upper extremity. In the cases of organic hemiplegia the onset was in the extremity or parts of an extremity relatively less affected by paralysis; a completely paralyzed part has been observed to be free from spasm. The jacksonian spasm is typically a clonic spasm, although circumscribed muscle spasm of tonic form has been observed. When the jacksonian spasm extends it progresses in such a manner that the muscles are progressively affected in the order of their cortical representation. Consciousness is retained or is only slightly clouded. Following the epileptic attacks there may be transitory aphasia (exhaustion palsy); also temporary motor paresis of a member (paralytic equivalent). Extra paroxysmal twitchings in certain muscle groups, which recall either voluntary movements, or choreiform and athetoid movements are occasionally observed. Epilepsia continua or long continued rhythmic muscular contractions, from six to eight in a second, was observed in one case.

In fifty of the cases a brain lesion was evidenced by sensory-motor symptomatology, and in forty-eight cases there was a more or less pronounced affection of the motor region. From the psychic side there were episodic changes of disposition lasting one or more days: irritability and sudden outbreaks of temper were characteristic. Posepileptic psychoses have not been observed in the series. Only occasionally characteristics of chronic epileptic character were observed—circumstantiality, littleness, pedantry, narrowness of the thought content and general interest, egocentric psyche, exaggerated politeness and religious tendency. Regarding the intelligence, memory attention and apperception were particularly affected; arithmetic was strikingly difficult, and fatigability or mental effort was marked. In the association tests delayed reaction time, missed reaction, tendency to perseveration (repetition of the test word), and sentence formation instead of word pairing were frequent phenomena. However, no striking differences were noticed in these traumatic epilepsies from the idiopathic forms, or severe head traumas uncomplicated with epilepsy.

Research directed toward the cardiovascular system corroborated the earlier observation of Goldstein that bradycardia was frequent, especially in the reclining posture; in one case the rate was found to be 36. Bradycardia was found, however, in cases of brain injury without epilepsy. The blood pressure as a rule was low—in the majority of cases below 130 (Riva-Rocci). The blood picture revealed a leukopenia before the attack and a leukocytosis following, lasting a number of hours and reaching a count of 16,500 in one case (five hours after the attack). This same change in the blood picture has been found in poisoning by Witte peptone and is characteristic of anaphylactic shock. This would suggest that the epileptic attack is accompanied by an intoxication of protein nature.

Regarding the question of disposition to attack, the author from his experience in these traumatic cases holds firmly to his former thesis that any brain will react by an epileptic attack, provided the damage or noxus be sufficiently intense. In twenty-nine cases in which a thorough investigation of the personal and familial hereditary history of the case was obtainable, only eight cases suggested possibility of an underlying predisposition in the affected person. Reflex epilepsy is excluded in the series; rather is the cause to be found in the brain lesion itself. The real problem in the traumatic epilepsy is to explain the cases in which the late attacks occur, namely, those cases in which an appreciable interval elapses between the trauma and onset of attacks. A small number may be explained by abscess formation or other local processes, such as plaques jaunes the result of trauma and contusion by blunt instruments. Nonpenetrating wounds by fracture may produce diffuse lesions of the brain causing general epileptic attacks.

The influence of the localization of the skull injury is discussed in detail with reference to available statistics which show that injuries of the parietal region, whether or not complicated by epilepsy, make up from 40 to 45 per cent. of all cases of skull injuries of warfare. In the author's series of epilepsy cases, however, there were thirty-seven cases, or a percentage of 63.7, with parietal wounds. In forty-two cases without epilepsy there were nineteen, or 45.3 per cent., with parietal wounds. The proportion of parietal cases with and without epilepsy would be 63.7 : 45.3. It is concluded that lesions of the parietal region of the skull have a particular signification for epileptic attacks, especially jacksonian attacks. As to the character of the lesion, it is particularly gutter, longitudinal and segmental wounds which are followed by epilepsy, as these wounds heal with large pulsating bone defects. In regard to the covering of defects, opinions vary greatly on this subject. Epilepsy is more frequently found in cases with than without bone defect, which is in part explained by the severe accompanying pathologic process affecting the brain substance itself.

When intracranial pressure is studied, bone defects are of especial interest. It is recalled that Kocher believed that epileptic attacks were caused by variation of intracranial fluid pressure and resulting disturbances of circulation in abnormally irritable cortical areas. A decompression operation with excision of the dura, forming a permanent valve, was advocated as a means of surgical treatment. Bier, however, by intermittent compression hyperemia by a neck band, thereby causing a variation of cerebrospinal fluid pressure, was not able to produce attacks in epileptics. He therefore refutes the Kocher hypothesis. Former investigations by the author in conjunction with Pötzl, of fluid pressure measurements in a considerable number of nontraumatic epilepsies, has not confirmed the contention of Kocher, although a certain significance of increased pressure is not denied. In twenty-six epileptic cases of this series lumbar puncture was done. In nine cases the pressure was within normal limits (120-150 mm. water); in eight cases between 150-180 mm.; in five cases between 180-200 mm.; in four cases over 200 mm. One patient, who later showed abscess formation, registered a fluid pressure of 160 mm. In eight cases in which an estimation of the protein content and cells was made, no deductions were made. In the author's opinion, Kocher's contention that a constant and significant increase of fluid pressure is present in these traumatic cases is not proved.

A survey of the German literature of war head injuries shows that considerable attention has been paid to the pressure and state of the cerebrospinal fluid. Tillman attaches considerable importance to general and local increase of fluid pressure as a cause of epileptic attacks. When the increase is in the subarachnoid space, stasis causes autointoxication of the brain by metabolic bye-products. Increase of fluid protein indicates inflammatory reaction.

Bungart believes that all gunshot head injuries are accompanied by a fluid pressure at or above the upper limit of normal. Payr associates meneingitis serosa with brain abscess, and thus explains alterations of the fluid. Hahn makes the following deductions: With normal pressure there is no danger of abscess; the brain defect may be covered. Increased pressure, but no protein increase, shows serous meningitis. These patients are prone to later epileptic attacks, and preventive surgical procedures to relieve pressure must be considered in order. Lumbar puncture alone does not permit a differentiation between encephalitis and abscess formation.

Puncture headache, backache and symptoms of meningismus, rigidity of neck and nausea were of frequent occurrence. These symptoms were also found following dry puncture. Two dry punctures were followed by attacks, as were also successful punctures. In a number of cases Bier's experiment of compression hyperemia was repeated with the result that the fluid pressure was somewhat increased, but no epileptic attack was produced.

A comparison of a series of gunshot injuries of the head shows that forty-four patients with epilepsy had splinters of bone or metal in the brain in 33.6 per cent., whereas in thirty-seven cases without epilepsy such splinters were present in 21.6 per cent. The presence of splinters, therefore, would not appear to be a determining factor in the attacks. Adhesions between the brain and its membranes, and the dura and the bone have been considered by many writers of most important pathogenetic moment, the effect being considered as mechanical and dependent, not on movement alone, but on each pulse beat, coughing and straining. When the injury does not affect the motor areas directly, necropsy cases have shown edema, cystic degeneration and inflammation extending to the motor areas as the most probable causes of attacks. It is thought by the writer that only by postoperative observation of the course of a considerable number of cases may the question of adhesion irritation and reflex action be decided.

The interval cases of epilepsy and the conditions that eventually cause attacks after weeks and months and even years after injury, are subjects of discussion. Encephalitis and meningitis epilepsy are compared. Summation of stimuli may be a factor dependent on increasing cicatrization, cystic formation, etc. Cases of head injury not developing epilepsy are finally considered. The author is inclined to believe that these patients differ from the epileptic patients in that the latter show a greater quantitative change in respect to the lesion, as well as a different localization.

The therapy of traumatic epilepsy is essentially surgical, although there is no consensus of opinion as to the indications or time of operation. The early results of operative treatment are always questionable, as one was accustomed to speak formerly of cure in one or two years, then of longer periods, until it is now held by some writers that a cure cannot be claimed within six years following operation.

A number of surgical procedures are described in the literature; excision of the cortical motor centers after Horsley; subcortical incisions of the same areas after Trendelenburg and covering in of the defect. In general, the last procedure has not been markedly successful. The clearing up of the wound by either primary or secondary operation, removal of splinters, opening of cysts and puncturing an edematous arachnoid are, of course, evident indications. The foreground of surgical interest has been occupied by severing and preventing recurrence of adhesions. Toward this end a fascia-flap has been transplanted in the dural defect, as suggested by Rehn. Koennecke has recently studied the results of this procedure histologically, and warmly recommends its adoption. A number of dura plastic operations have been devised and the method of Finsterer has received especial attention. This consists of reconstructing the subdural and subarachnoid spaces and inserting a piece of prepared hernial sac so that it will extend beneath the edges of the dural defect.

The author's patients were operated on by a number of different surgeons, many of the operations being early ones for the removal of splinters, foreign bodies and for the purpose of removing dead tissue and cleansing the wound. Many late operations for abscess cyst formation, for adhesions and for defects of bone and dura were also performed. In no case was a persisting successful result as regards recurrence of attacks obtained. The same experience has followed drug therapy, luminal and bromids.

The procedure of Spielmeyer—application of cold by means of an icebag for one-half hour twice daily over the lesion—has been tried by Podmaniky without success.

The article is a comprehensive one of 110 pages, the literature references are numerous both in text and bibliography. The case histories of thirteen cases with illustrations follow the main text. The article may be considered representative of the experience of the central allies in traumatic epilepsy.

Schaller, San Francisco.

PROGRESSIVE SUBACUTE FOCAL LEUKO-ENCEPHALITIS. Henri Claude and Jean Lhermitte, L'Encephale **15**:89, 1920.

The authors borrowed the term leuko-encephalitis from Rémond (of Metz) who used it to explain the pathogenesis of a systematic delirium which he thought was due to the destruction of certain fibers of the white matter of the cerebral hemispheres. His conception was hypothetic and not confirmed by an anatomic examination.

Claude and Lhermitte have applied the term leuko-encephalitis to a disease which they have been able to diagnose prior to death and which is caused by disseminated foci of encephalitis in the white substance of the hemispheres and the corpus callosum, involving also the central nuclei but in no way involving the cortex. These foci develop subacutely and are characterized by alterations of anatomic elements which are quite distinct from those observed in acute encephalitis, the softenings, the scleroses of the multiple sclerosis type, the scleroses of syphilis or the encephalitis of infancy. Finally, the authors state that the progress of the disease is sufficiently characteristic to warrant considering it an anatomic and clinical entity.

The description of a new disease entity is based on a long clinical and pathologic report which the authors summarize thus:

A young man of 20 was admitted to a hospital for an excoriation of the foot due to irritation by his shoe. He was without any appreciable hereditary or acquired disease. He began to suffer at the time of admission, i. e., in the month of August, 1915, from weakness of the lower extremities. This increased so that at the end of three months it was quite impossible for him to walk. This paraplegia, which was accompanied by complete absence of the tendon reflexes and by marked signs of incoordination, soon diminished. Then it became worse so that the patient was completely bedridden on Dec. 15, 1915. At this time the clinical picture was quite different from that of October for there was present a marked contracture of the lower extremities, and an almost complete paralysis of the upper extremities. Moreover, the patient's mental state, normal until then, became abnormal in that he sank into a stupor and was incapable of response. Finally, the sphincter control was lost.

Just after this had occurred, a marked improvement began. He emerged from the stupor on Jan. 10, 1916; voluntary movement returned in all the extremities, and in February the patient was normal mentally, was able to get up, walk and execute all movements of the upper extremities. A rapid convalescence was expected. This expectation was not realized as on April 20, 1916, fever appeared at the same time as a return of the paralysis of the extremities and the patient's speech became so confused that it was incomprehensible.

On May 15 there was great improvement. The patient was able to stand up and walk with the aid of two canes and two nurses. The movements of the lower extremities were poorly coordinated. A month later (June) a complicated group of symptoms was observed which included aphasic phenomena, that is, alexia, agraphia and apraxia. The examination showed that speech disorders as well as those of voluntary movement (apraxia) were completely independent of all sensory disorders of function. Not only was no anesthesia demonstrated but examination of the fundi was completely negative.

Except for the appearance of vomiting of cerebral type and of some unsystematized delirious ideas, the patient's state remained about the same until his sudden death January 10. The authors add that, although there was no further improvement as marked as that recorded above, nevertheless the psychic and aphasic manifestations showed remarkable oscillations. The spinal fluid was negative in every way.

The alteration of periods of aggravation and improvement constitute one of the most striking features of the case above summarized. It was due to this special form of evolution and to the absence of all symptoms signifying a cranial hypertension (absence of choked disk and of increased pressure of the spinal fluid measured by Claude's manometer) that the authors were able to reject the hypothesis of brain tumor and to make the diagnosis of central encephalitis, involving the centrum ovale and the corpus callosum. The anatomic examination proved that this diagnosis was correct.

The necropsy findings summarized were: There were lesions of the centrum ovale, corpus striatum and optic thalamus. The cerebellum, spinal cord, medulla, pons and midbrain were negative except for secondary degeneration of the pyramidal tract. The cortex was normal. The lesions were greatest in the centrum ovale and radiated toward the gray covering on one hand, toward the basal ganglions on the other. The lenticular nucleus and the optic thalamus were most affected. The lesions were limited by the ependymal epithelium, below which the ventricular neuroglia proliferated abundantly. In the centrum ovale, the lesions extended to the corpus callosum and the white commissure and the lesion was continuous from one side to the other. Neither the meninges nor the blood vessels were diseased.

The minute changes were of two types, inflammatory and necrotic. The former were shown by round cell infiltration in the perivascular space and in the walls of the vessels. The neuroglia was greatly increased. There were astrocytes and large giant cells with thick processes. The latter, the necrosis, showed as distinct destruction of small portions of the cerebral parenchyma in spots of varying size. The vascular lesions were not all of the same age. In some places the round cell infiltration was most conspicuous, while in others the process was older, there being vascular sclerosis and hyaline degeneration. In some vessels obstructing thrombi were seen. All intermediary stages were found. This thrombotic process accounts for the areas of necrosis described.

The authors believe the process is of toxic infective origin and characterized by a vascular inflammatory reaction leading to thrombosis which in turn leads to necrosis of the various parts of the brain mentioned above.

They review the literature briefly and state that but one case corresponds to this one, namely, a case reported by H. Claude and P. Lejonne. In this case no necropsy report confirmed the diagnosis.

Claude and Lhermitte emphasized particularly the remissions as forming a prominent characteristic of the disease.

Kraus, New York.

STUDY OF ADMISSIONS TO SING SING. Bernard Glueck, Ment. Hyg. 2:85 (Jan.) 1918.

Criminology should seek in psychiatry an end toward the definition and administration of its problem because:

1. Of 608 adult prisoners studied by psychiatric method, 66.8 per cent. were not merely prisoners but persons who had shown through life a tendency to behave in a manner at variance with the behavior of the average normal person, and this deviation from normal behavior had repeatedly manifested itself in a criminal act.

2. Of the same series of 608 cases, 359 or 59 per cent. were classifiable in terms of deviations from average normal mental health.

3. Of these 359 deviations, 171 or 28.1 per cent. of the 608 persons studied possessed a degree of intelligence equivalent to that of the average American child of 12 years or under; of ninety-eight native born defectives, 80.6 per cent. were recidivists in crime.

4. Of the 359 cases, 115 or 18.9 per cent. of the 608 were constitutionally inferior or psychopathic.

5. Of the 359 cases, 73 or 12 per cent. of the 608 were suffering from distinct mental diseases or deteriorations.

It is inferred from these fact that one is dealing with a highly selected and highly specialized group of human beings and to the student of behavior a knowledge of the person back of a given act is absolutely essential if a clear understanding of the nature of behavior is to be had. No agency concerned in the administration of this problem must lose sight of the common goal or end, namely, the necessity for readjustment of that badly adjusted person, the criminal; but in emphasizing the constitutional factors, the psychiatrist must not ignore many environmental factors which contribute directly or indirectly to commission of crime.

These criminals may be divided into the following classes:

Classification.—(1) intellectually defective, (2) mentally diseased or deteriorated, (3) psychopathic, (4) unclassified (epileptic).

General Statistical Considerations.—Of 608 cases, 359 or 59 per cent. were classified as follows:

	Number.	Per cent.
Mentally diseased or deteriorated	73	12
Intellectually defective	171	28.1
Psychopathic or constitutionally inferior	115	18.9
	359	59
Type of offense:		
Crimes of acquisitiveness	388	63.8
Crimes of pugnacity	148	24.3
Sex crimes	60	9.9
Perjury, dynamiting, arson, aiding escape of prisoner, abandonment	12	2
	608	100.0

Thus, frequency of a given type of offense is in inverse ratio to its seriousness, and extent of psychopathologically classifiable cases is in direct ratio to the seriousness of the offense, thus:

Acquisitive crimes psychopathologically classifiable	57.2
Crimes of pugnacity psychopathologically classifiable	58.8
Sex offenses psychopathologically classifiable	70.0

Recidivism.—There are two methods in estimating the degree: (a) frequency with which the individual had come in contact with the law; (b) history of previous sentences to penal or reformatory institutions. In this study the latter method is used. Of 608 cases, 406 or 66.8 per cent. had served sentences before. The factor of nativity was extremely significant, for in the foreign born, recidivism was 49.8 per cent.; in the native born, recidivism was 75.9 per cent.

This decidedly lower percentage of recidivism in the foreign born is undoubtedly due to the fact that accidentally environmental factors play a much greater rôle in the foreigners' conflict with the law.

1. *The Defective Group.*—171 cases or 28.1 per cent. of the 608 cases studied were diagnosed as intellectually defective. In arriving at the diagnosis of mental deficiency, the current psychometric methods were used, Yerkes-Bridges point scale, Terman revision of Binet-Simon test, and the Healy construction test; the diagnosis does not rest solely on laboratory findings, but takes into account also the actual capacity for adaptation which each person manifested in his past life.

Notwithstanding the urgency to do everything possible to readjust the adult offender at the time of his contact with a state prison, the time for active effort in the majority of instances was in the past, especially during their contact with public schools.

The 171 cases comprised all of the defectives among the 608 cases, but as sources of information in cases of foreign born were incomplete, the detailed consideration of the problem of defectiveness will be confined to native born, ninety-eight cases or 57.3 per cent. of the defective group. Of these ninety-eight, the majority fall in various grades of moronism. These findings are particularly significant, as they demonstrate that the higher grade of defective constitutes the most serious problem in antisocial behavior.

Recidivism: Under this head were included seventy-nine cases out of ninety-eight, or 80.6 per cent. Compared with the general percentage in 608 cases, 66.8 per cent., we have an increase of 13.8 per cent., a rather significant correlation between mental defect and tendency to antisocial behavior.

Heredity: Hereditary factors of some significance came to light in forty-eight of ninety-eight cases—principally alcoholism and tuberculosis.

There were early pathologic manifestations in fifty-two instances, such as running away from home, delayed nocturnal enuresis, stuttering, sleep walking, early criminality, hysterical seizures and convulsions. These manifestations might have been noticed had the persons been reared in the proper environment. In twenty-five cases evidence came to light of association in early life with criminal and antisocial types, and in many instances they were deprived of parental supervision. Failure to appreciate these indications of a pathologic state during the school career cannot be easily condoned. At that time some provision should be made for placing these persons in a more appropriate environment.

2. *Mentally Diseased or Deteriorated Group.*—Seventy-three cases or 12 per cent. of the 608 were classified as follows: arteriosclerosis, 3; morphin, 1; dementia praecox, 36; manic-depressive psychosis, 2; markedly cyclothemic, 1; paranoid state, 3; syphilitic, 13; alcoholic, 14. Total, 73.

Recidivism: Of seventy-three, forty-six or 63 per cent. were recidivists. As has been stated, 406 or 66.8 per cent. of the 608 were recidivists. Therefore, as far as mental disease is concerned, the problem of habitual recourse to antisocial behavior is not an especially significant one as compared with the defective and the psychopath.

3. *The Psychopathic or Constitutionally Inferior Person.*—One hundred and fifteen or 18.9 per cent. of the 608 criminals were thus classified. Diagnosis was based on a study of the life history of the person and his mode of reaction to various environmental influences. In addition, certain laboratory tests were carried out which aided in demonstrating the high degree of suggestibility and emotional instability that are so characteristic of the psychopath. Here again only native born are considered. Of 115 cases, ninety-one or 79.1 per cent. were native born.

Recidivism: Of ninety-one, seventy-nine or 86.8 per cent. were recidivists, a higher percentage than in any other group and 20 per cent. higher than in the 608 cases; therefore, the psychopath has contributed in the largest measure to this group of *habitually* antisocial people. For the seventy-nine recidivists in this group the average number of sentences per man is 3.9, although the most requent chronologic age in the 608 cases was only 23 years.

FOREIGN GROUP.—Of 608 cases, 213 or 35 per cent. were foreign born, Italy and Russia contributing the most significant share. The average foreigner was committed at a later age than the native born—the foreigner at 27 years, the native at 22.

Recidivism: Only 49.8 per cent of foreigners came under this head as compared to 75.9 per cent in the native born. Foreigners may be called "accidental offenders," adverse social and economic conditions frequently adding to the chances of committing an offense.

In estimating intelligence in the foreign born factors of education, length of residence in the United States, age at arrival, and language factor involved in intelligence scale were taken into consideration.

Italian Group: This comprised sixty-eight cases. Forty-three persons or 63 per cent. were mental anomalies. The high percentage of illiteracy and meager education of those who did attend school, accounts in a measure for the general low level of intelligence as shown by the intelligence tests. Crimes against person (other than sex), which derived their impulse from the instinct of pugnacity, constituted the largest proportion of all offenses, 48.6 per cent. The economic level was low; recidivism: 22 cases, 32.4 per cent.

Russian Group: Sixty-eight per cent. were Jews. There were fifty-eight cases—thirty-one mental anomalies, or 53.4 per cent; recidivism, 66.3 per cent. Most frequent crimes were those having impulse in instinct of acquisitiveness. Level of intelligence was higher than in the Italian group; but the percentage of recidivism was doubled; with this increase in recidivism was found a more or less corresponding increase in unemployment and idleness at the time of arrest.

German Group: This comprised twenty-five cases. Eight or 32 per cent. were mental anomalies. Recidivism was high—68 per cent. In this matter it is hard to draw any general conclusions, for this group shows on the one hand the lowest percentage of psychopathologically classifiable cases, and on the other hand represents perhaps the group of highest culture. There was a predominant tendency to crimes of acquisitiveness.

Austrian Group: This includes eighteen cases—thirteen or 72 per cent. were mental anomalies—the largest percentage of all; recidivists, 55 per cent.

Miscellaneous Group: (Irish, Greeks, English, French, Dutch, Danish, etc.): This included forty-four cases. Twenty-nine or 66 per cent. were mental anomalies; twenty-two or 50 per cent. were recidivists.

General Conclusions from Study or Foreign Group: 1. Of 608 cases, 213 were foreigners, 35.3 per cent. In the counties from which Sing Sing derives its prisoners, there were 52.2 per cent. more foreigners than native born.

2. Of 213 cases, 124 or 58.2 per cent. were mental anomalies. This is significant when we remember that the majority of these cases were excludable at the time of their arrival in the United States.

3. The immigrant is less likely to commit crime than native born. Recidivism is 49.8 per cent., compared to 75.9 in natives.

4. Immigrants showing higher level of intelligence tend to crimes of acquisitive nature; those of lower levels are guilty of crimes against person and sex crimes.

5. Under the new Immigration Law, forty-four of 213 would have been excluded on account of illiteracy, and thirty-five others on account of coming in contact with the law within five years before arrival in the United States.

6. There is a certain correlation between the degree of recidivism and the extent of unemployment at the time of the commission of the crime.

7. There is no correlation between degree of psychopathologically classifiable cases and degree of recidivism in immigrant groups.

8. There is a slight tendency to naturalization.

Activities of the Clinic.—(a) The clinic should make a delineation of the debits in the prison's make-up which might have been responsible for bringing him in contact with the law. (b) It should outline the type of credit, or the desirable qualities the person may possess, the cultivation or development of which will tend to balance debit side. For this purpose the following plan suggests itself:

There should be a new prison at Sing Sing to serve as a classification and research station for the entire prison system of the state. It is intended to keep each prisoner under close observation and study and under intensive vocational training for such a period to define clearly the problem which he presents—perhaps for three or four months. The problem of the adult prisoner is concerned, then, with five more or less well defined types of persons which the various penal institutions of the state should receive on their discharge from the clearing house.

1. Normal young adults capable of learning a trade should be sent to either of two industrial prisons—Dannemora or Auburn.
2. Older normal prisoners should be sent to the agricultural prison at Great Meadows.
3. Insane delinquents should be sent for permanent care to Dannemora State Hospital; for transitory disturbances, to a psychiatric pavilion at a reception prison.
4. The defective delinquent should be sent to an institution for more or less permament segregation.
5. The psychopathic delinquent—treatment should be determined after a thorough investigation by a competent Board of Inquiry.

SCHALLER, San Francisco.

LOCAL PARALYSIS FOLLOWING SUPERFICIAL INJURIES NOT INVOLVING NERVE TRUNKS; WITH SPECIAL REFERENCE TO TRAUMATIC "ASCENDING NEURITIS" AND TO "REFLEX PARALYSIS." F. M. R. WALSHE, Brain **42:**339, 1920.

The author discusses a group of cases of local paralysis, following trivial superficial injury to the distal extremity of the limb, in which no nerve trunk was involved yet which presented the symptoms of an organic nerve lesion. He suggests two causes to explain the symptoms: (1) "reflex paralysis" and (2) "ascending neuritis." The concept of reflex paralysis is now almost abandoned and is accepted only in cases of arthrogenic paralysis, such as have been reported by Oppenheim and Kramer. He suggests that the ill defined and heterogeneous collection of clinical forms that Babinski has named "reflex nervous disorders" is not a true clinical entity but rather a medley of abnormal conditions to which hysteria and mechanical effects of tissue changes and even local tetanus may contribute their share. He then discusses the alternative explanation, ascending neuritis, which Sicard has recently described in a most comprehensive study. The latter regards the condition as exceedingly rare. He defines it as a localized, slowly progressive lesion, ascending from the distal terminations of the nerve toward a central end. It is constantly associated with an infection of a cutaneous lesion in which nerve endings are affected. In a careful review of the clinical and pathologic studies of French and German literature, he finds that obscurity pervades the whole subject. He states, however, that there is sufficient clinical evidence that the syndrome of "ascending neuritis" is not an example of a "lymphogenous toxi-infective" process as described by Orr and Rows. Four cases presented by the author correspond in their clinical picture to the syndrome of ascending neuritis:

CASE 1.—This consisted of a small suppurating wound of the dorsum of the foot followed by paralysis of all muscles supplied by the external popliteal division of the sciatic nerve with corresponding sensory loss.

Case 2.—A superficial wound of the lower third of the thigh was followed by paresis and wasting of the quadriceps extensor femoris and diminution of the knee-jerk.

Case 3.—The patient received a penetrating wound of the leg in its distal third which resulted in drop-foot and diminished faradic excitability of all muscles below the knees. There was no sensory loss; but there were diminished ankle-jerk, cyanosis, coldness and lesions resembling chilblains on the foot.

Case 4.—A blow received on the heel caused no wound. Severe and progressive atrophic flaccid palsy of all muscles supplied by the sciatic nerve followed with a corresponding change in the reflexes, and sensory loss. There were marked cyanosis and coldness of the foot.

The septic factor is excluded. In conclusion he states that in the absence of any sound pathology of these clinical conditions it is impossible to dogmatize as to the underlying lesion. They indicate, however, that in rare instances the clinical manifestations of a local neuritis may follow trivial injuries commonly associated with organismal infection of the afferent terminations of the nerve affected.

Blitzsten, Chicago.

THE SOMATIC CAUSES OF PSYCHONEUROSES. Charles L. Dana, J. A. M. A. **74**:1139 (April 24) 1920.

Dana deplores the emphasis placed on the psyche as the sole etiologic factor in the psychoneuroses. His argument is based on the theory of "synapsis," which implies "breakable points and points of variable resistance in the finer organizations of the brain." Here the traveling nerve impulse meets its highest resistance and neural phenomena are apt to develop. Paths of conduction may be blocked or made free not only by mental but also by physical agencies and commotional changes, endocrine disorders, toxemias of various kinds, etc., may all act as etiologic agents. A second hypothesis is presented, and the effect of concussion, as an instance, is traced through the stages of physical changes in the nervous system conditioned by molecular changes, the emotion, the subconscious desires with defense and adjustment activities, the organic or biochemical nerve disturbance and finally the fully developed psychoneurotic phenomenon. It is asserted that the steps of emotion and subconscious desires may be absent altogether and that in nontraumatic neuroses the affect reaction and the organic or biochemical processes are dominant. In any event, there is actual material change in the nervous system. These neural modifications are, of course, temporary and may be adjusted by a mental effort or suggestion treatment.

Dana is theorizing, but his theories are ingenious and interesting and do not wholly lack scientific support. For instance, Carver and Dinsley's experimental research (Carver, Alfred, and Dinsley, A.: Some Biological Effects Due to High Explosives, *Brain* **42**:113 [June] 1919) showed paralyses and nervous disturbances in the fish and rats placed in the zones of indirect concussion. The same observations were made on men working in demolition stations protected from direct concussion and presumably not subject to fear.

It has been the reviewer's experience, that almost 50 per cent. of the cases diagnosed as psychoneuroses of various types, and presenting the symptomatic picture of so-called functional neuroses, revealed on careful and extended physical examination the presence of such conditions as anemia, arteriosclerosis, diabetes, endocrine disorders, nephritis, pelvic disease, pyorrhea, apical abscesses, syphilis, tuberculosis, etc. It may be too much to assume a direct causal relationship to the neurosis in every instance, yet such definite pathologic states cannot be ignored. An energetic effort to correct these underlying physical defects will often result in recovery from, or marked improvement in, the neurosis.

STRECKER, Philadelphia.

CONCERNING PRISONERS. BERNARD GLUECK, Ment. Hyg. **2**:117 (April) 1918.

There is today a serious lack of coordination of effort in criminal administration. The notion of "born criminal" is fatalistic in its finality. The statement that persons by the very act of their recidivism have given proof of being irreformable, either as born or instinctive criminals or as otherwise predestined to their careers, can no longer satisfy the mind of any one who approaches the situation without bias. As soon as the person is put in confinement he is despaired of, his past ceases to be of interest, his future ceases to be viewed as a problem containing unknown possibilities. It is precisely at this stage that the need arises for close observation and open-minded study of his behavior both past and present if treatment and eventual discharge back into the community is to have any helphul bearing on his future course.

The danger lies in ignoring the medical definition of the man back of the act. There is a fallacy in trying to fit the sentence to the criminal act instead of to the criminal back of the act. The man whose constitutional disease expresses itself in antisocial behavior on the one hand and in disordered mentality on the other, has not received adequate attention when the one phase of his difficulty is dealt with independently by the imposition of a definite sentence, and the other phase by temporary treatment in a hospital for the insane for the period of the acute manifestation of his mental disorder. In either case treatment has been directed to an attribute of his diseased personality and not to the personality as a whole in its relation to environment. The majority of psychopathic persons show in childhood and early boyhood unmistakable signs of pathologic personality. It is in the early formative period, especially during boys' contact with the school system, that a better public appreciation of these danger signs is needed and a disposition to institute the proper preventive measures; there is also necessity for recognition that there is a great problem in understanding the causes and nature of delinquency; there is a necessity for a revision of procedure in the direction of individualization. The community must know: 1. That the surest protection against the spread of crime is effective handling of the criminal. 2. That such knowledge must proceed from a detailed knowledge not merely of his guilt in a given instance, but of himself, his history, his fixed limitations and his possibilities for rehabilitation. 3. That the test for efficiency in a modern penal system is its success in turning out not model prisoners, but decent citizens.

SCHALLER, San Francisco.

NOTE SUR LES CANALICULES INTRACELLULAIRES DES CELLULES NERVEUSES (NOTE ON THE INTRACELLULAR CANALICULI OF NERVE CELLS). J. CARAMANIS, Schweiz. Arch. f. Neurol. u. Psychiat. **5**:264, 1919.

Nelis, Holmgren and Joris have shown that the protoplasm of nervous cells is traversed by delicate canals, which occasionally form a sort of network, to which Nelis gave the name spireme, and Holmgren, the name of reseau canaliculaire. This is present in all the cells of the cerebrospinal axis and stands in intimate relation with the tigroid bodies. Holmgren and Pugnat thought that this network was connected with the pericellular lymphatic spaces; more recently Holmgren has expressed the opinion that they represent a liquefaction consequent to penetration by neuroglia cell processes, the purpose of which is to serve the nutrition of the cells. According to Legendre this hypothesis is reversed, the glia cells in question playing the rôle of phagocytes. According to Golgi, who used the silver method, these canals represent an intracellular structure. Cajal and Holmgren look on the canals and the reticulum as identical structures, while von Bergen endeavors to distinguish the two.

The author employed the toluidin blue stain, according to the Lenhossek technic. These observations were carried on altogether on the motor cells of the anterior horns of an adult cat, which died of starvation at the end of eight days, and which had otherwise been well. The writer believes that the structures under consideration are formed, not by an intracellular network, but by intracellular canals. These canals present markedly varying configurations, have no characteristic localization, occupy both the periphery and center of the cell, proceed in any direction, even through the nucleus, and anastomose in an intricate manner. Some of these canals are seen to open on the surface of the cell. They do not appear to have an independent wall, but are surrounded by granules derived from the chromophil substance. It is striking, indeed, that these canals are in intimate contact with the tigroid bodies, a finding that convinces Caramanis that there is a functional relation between the two. He is of the opinion that these canals are formed at the moment that the protoplasm disintegrates and probably represents a dissolution of the chromophil substance rather than of the spongioplasm. The formation of the canals is, therefore, the result of an accidental elimination and indicates definite cell pathology. The canals represent the initial stage of cavity formation, which may progress to complete cellular disintegration.

Woltman, Rochester, Minn.

VACCINE THERAPY IN CEREBROSPINAL MENINGITIS. Lucien Girard, Bull. méd. **34**:313 (March 27) 1920.

The proper therapy of cerebrospinal meningitis due to the meningococcus, the author says, is a combination of serum and vaccine therapy; he gives several reasons for this. Serum alone is inefficacious when the infecting germ is atypical and no specific serum is at hand, when the infecting germ is resistant to the serum, when there is a meningococcemia, and when the meningitis has entered on a chronic phase with or without localization of the infection in the middle ear, or elsewhere.

Nine cases are cited (reported by others) in which cure was obtained by vaccine therapy. An autovaccine is presumably not considered superior, though the results thus far reported have been obtained largely with autovaccines only because stock vaccines are not easy to procure. That prepared by Sergeant and Bordet is the only one at present (in France).

These points should be added regarding dosage and administration: Boidin and Weissembach commence by injecting 350 million organisms subcutaneously. Florand and Fiessenger make an initial injection of 1,000 millions, while Mery and L. Girard (the writer) begin with only 200 millions. The doses are gradually increased until 2, 3, 5 or 10 billions are reached. The injections are given at intervals of three or four days or a week. Recently, Sergeant and Bordet report the intravenous use of a stock vaccine in a meningococcemia (with cure).

Lewkowicz has shown that under the influence of the vaccine the serum of the patient acquires immune properties and the titer for specific antibodies becomes equal to that of the therapeutic serums of commerce.

Davis, New York.

OCULAR ATAXIA OF TABETICS. A Cantonnet, Presse méd. **28**:156 (Feb. 25) 1920.

Paralyses and pareses of extrinsic eye muscles in tabes are contrasted with ataxias in a study of cases reported by various observers. These ataxias—disorders of the associated movements of the eyes—are uncommon, but have been found either in the presence or in the absence of ataxia of the extremities.

Ocular ataxia may be evident on the simplest observation, appearing as an irregular and independent stopping and starting of the two eyes when an attempt is made to follow a finger moving from side to side; or it may be latent. A simple means of demonstrating this is quoted from Miraillé and Declaux: A truncated cone of paper 25 cm. long, rolled to 4 cm. in diameter at the proximal end and 1 cm. at the distal, is placed before one eye of the subject, who fixes an object 5 meters distant. If unstable binocular vision is present, diplopia appears when the tube is in place and disappears when it is removed, since the axis of vision of one eye is fixed and the other eye permitted to drift. This procedure shows simple eye muscle palsies as well as incoordination, but a differential diagnosis can be made by observing the direction and degree of diplopia, which are fixed in paralyses and variable in ataxias.

Huddleson, New York.

A PROPOS DES GRIFFES (POSITION OF FINGERS FOLLOWING NERVE INJURIES). M. Gaston Giraud, Progrès méd. **35**:113 (March 13) 1920.

The writer has contrived a means to express by formula the position of the fingers in the various hand positions found in peripheral nerve injuries. In his scheme each finger is represented by a roman numeral, the thumb being I, in each finger each phalanx is expressed by an arabic numeral, 1 applying to the proximal phalanx, and the capital letters "F" and "E" stand for flexion and extension, respectively. A plus sign occasionally used explains itself. How these terms are to be made into a formula is perhaps clear when it is stated that the writer formulates the attitude of the normal hand thus (IV-V) $F_1F_2F_3$. As a further example, in the case of an ulnar lesion in the forearm, because of the loss of function of the interossei and lumbricals, the resultant attitude requires the formula (IV-V) $E_1F_{2+}F_{3+}$. This much gives the method.

Formulas are given for many situations—for several subgroups under each of his three main classes, which are: (1) atonic hand attitudes; (2) hypertonic hand attitudes, and (3) hand attitudes with contractures.

This sort of work is not lacking in scientific accuracy, but it is academic and hardly of practical benefit. Such a method could not be used from a book by some one not already able to think in terms of the nerves, muscles and joints involved; and, on the other hand, any one so trained would arrive at the diagnosis without the aid of so mechanical a procedure.

Davis, New York.

A SIMPLE METHOD OF DISTINGUISHING WHITE FROM RED CELLS IN SPINAL FLUID CELL COUNTS. S. R. Gifford, J. A. M. A. **74**:1024 (April 10) 1920.

In the procedure employed by Gifford, the fluid is taken in two test tubes. To one of these, containing about 2 c.c., is at once added one drop of Loeffler's methylene blue, and the tube shaken gently. The second tube is saved for other tests. Examination after two minutes reveals the nuclei of the white cells staining distinctly so that they may be easily distinguished from red cells by low power, and lymphocytes from polymorphonuclears by high power. If the stain does not act quickly, two or more minutes may be required, but never long enough for coagulation to affect the results appreciably, except in a very heavy, turbid fluid.

Klauder, Philadelphia.

MYOCLONIC ENCEPHALITIS. J. A. Sicard, Presse méd. **28**:213 (April 14) 1920.

Written Feb. 6, 1920, this contribution takes up five cases in detail, observed during the preceding three months; up to that time no cases of encephalitis lethargica with myoclonic symptoms had been reported (in France), but a note appended to the article cells attention to the large number reported in the interim, from February to April. It is stated that some observers believed the myotonic to be more frequent than the ocular or lethargic manifestations. Transitional cases had also been reported.

The cases described here were characterized by a week or ten days of severe cervical, brachial and intercostal neuralgic pains, with moderate fever, followed by a period of about the same length or less when these pains disappeared and myoclonic contractions set in. During the third week, delirious symptoms were apparent, progressing slowly to coma and exitus; or else, a slow improvement appeared about the third week and recovery took place. The contractions typical of these cases were generally strong, rhythmic, occurring every minute or two, in one or a group of muscles, and showing a predilection for abdominal wall and diaphragm. Other findings were characteristic of encephalitis lethargica as earlier described.

Huddleson, New York.

ALIMENTARY ANAPHYLAXIS DUE TO PANCREATIC INSUFFICIENCY. M. Nathan, Bull. méd. **34**:59 (Jan. 21) 1920.

The writer recounts a case that offered convincing clinical confirmation of the experimental work of Lesne and Dreyfus. These workers have shown that egg albumin subjected to the action of gastric juice, retains its ability to cause an anaphylactic reaction when injected into a sensitized animal, but that the anaphylactic reaction is absent if the egg albumin is subjected to the action of pancreatic juice before injection. His patient was a child of 8 years who had always shown marked (intestinal and skin) anaphylactic reactions to ingestion of egg albumin in any form. The child was normally developed and examination of the abdomen showed only a slight distention. However, analysis of the feces showed pancreatic deficiency (numerous undigested meat fibers). The child was given 40 centigrams of pancreatin a day and while taking this, egg was added to the diet successfully. Several times the pancreatin was suspended and immediately there was a return of the anaphylactic reaction. The latter feature excludes the possibility that the method of giving the egg albumin (in small increasing amounts) had been responsible and makes clear that the pancreatic feeding was the essential thing. The writer believes that every case of anaphylactic reaction to egg albumin should be examined for possible pancreatic deficiency, either by the Cammidge method or by the examination of the stools, and believes that prolonged, perhaps permanent, feeding of pancreatin will be the treatment of choice.

Davis, New York.

GENERAL ABSENCE OF TENDON REFLEXES IN CASES WITH WOUNDS OF THE CRANIUM INVOLVING THE MENINGES.

A. Souques, Méd. **1**:274, 1920.

Souques believes that this symptom has often been overlooked. He believes it due to pressure on posterior spinal roots by spinal fluid at increased pressure. He found increased pressure and increased albumin in cases of trauma three or four months after the injury. This he believes due to mild infectious meningitis which passed unnoticed, and of which the increased pressure and albumin were the only evidence.

He cautions against mistaking areflexias of this kind in nontraumatic cases for tabes dorsalis.

Kraus, New York.

PSEUDOHYPERTROPHIA MUSCULARE DE LA JAMBE CONSECUTIVE A UNE BLESSURE DU NERF SCIATIQUE (PSEUDOHYPERTROPHY OF THE LEG FOLLOWING A WOUND OF THE SCIATIC NERVE).

L. Cornil, Progrès méd. **35**:117 (March 13) 1920.

In a case seen twenty-eight months after injury (a buttock wound by a bullet) there was a clinically incomplete interruption of the internal popliteal with slight irritative painful phenomena, fibrillations, impaired (returning) flexion of toes and foot, tactile and pain hypesthesia in the zone of the posterior tibialis, partial reaction of degeneration in the zone of the internal popliteal, abolition of the ankle and medioplantar reflex, and as the striking sign a hypertrophy of the calf muscles on the affected side. Subcutaneous edema and thickening of adipose layers were not present to explain the finding nor were there circulatory modifications. The muscle mass itself gave a marked feeling of elasticity. The author reports the case as one showing "true interstitial muscular pseudohypertrophy" dependent on nerve alterations, and mentions that two similar cases have been reported by Lhermitte and another by Tinel.

Davis, New York.

FIFTEEN CASES SHOWING INVOLUNTARY MOVEMENTS FOLLOWING ATTACKS OF GRIP AND LETHARGIC ENCEPHALITIS.

Pierre Marie and G. Levy, Méd. **1**:270, 1920.

This is an account of myoclonias of the head and extremities appearing during epidemic encephalitis. It is merely a review of these associated clinical manifestations accompanying the disease. They differ in no way from what is commonly found. This review is interesting.

Kraus, New York.

Society Transactions

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Feb. 27, 1920

S. D. Ingham, M.D., *President*

AN UNUSUAL CASE OF OCCLUSION OF THE POSTERIOR INFERIOR CEREBELLAR ARTERY. Presented by Dr. N. W. Winkelman.

A man, aged 54, whose family history was negative, denied syphilis, but admitted that he drank to excess. On Sept. 28, 1918, at 8 a. m., while waiting for a car, he had severe occipital pain, became unsteady, staggered, but reached home without assistance. He could not speak above a whisper and had much difficulty in swallowing. He remained in bed a month because of unsteadiness in walking. The left side of his body felt as though asleep. At present he has a tendency to stagger and heat applied on the left side of the body gives a sensation of tingling.

Physical Examination. — The base of support was widened, and he was unsteady with eyes open or closed. The right pupil was smaller and the right palpebral fissure was narrower than the left; a slight right enophthalmos was noted. There was no nystagmus; the uvula was drawn up and to the left. The motor system was normal.

Sensory Examination.—Sensation of the face was normal. Sensation to touch of the body and extremities was normal. Sensation of pain, heat and cold was markedly lessened on the left upper and lower extremities and the left half of the body. The pharyngeal reflex was absent. All other reflexes were normal. The blood pressure was: systolic, 152; diastolic, 82. The Wassermann and the urine tests were negative. Bárány tests were made. The lesion was located in the pontile region. The peculiarity in this case was the non-involvement of the descending root of the fifth nerve. It is the first case of its kind in the literature.

A CASE OF BRAIN TUMOR WITH WERNICKE'S HEMIPUPILLARY INACTION SIGN. Presented by Dr. Sherman F. Gilpin for Dr. F. X. Dercum.

J. D., a boy, aged 9, was admitted to the Jefferson Hospital, Feb. 6, 1920. He had been suffering from vomiting, some disturbance of vision and headache. The family and personal history were negative.

Present Illness.—Three months ago the patient began to vomit and had a slight headache. For two weeks he vomited about every other day; then the vomiting increased to about twenty times a day. Severe temporal headaches accompanied the vomiting. After a vomiting spell, which sometimes lasted from six to eight hours, the patient was hungry and retained food. Spells of such severity occurred every few days.

Just before Christmas the eyes became crossed. This condition has persisted. Two weeks ago, during a spell, he said he could not see and acted queerly. After a spell is over he does not remember anything about it.

He vomited from midnight of February 4 until the following forenoon; he screamed continuously and was difficult to control. February 6 he was apparently all right again, and examination showed slight weakness of the left arm and leg; face normal. There was slight incoordination of the left arm with adiadokocinesis and asynergia of left leg. Gait and posture showed a slight tendency to spread the legs. The reflexes and sensation were normal. There were choked disks with white star-shaped figures in the macular region and retinal hemorrhages. Left homonymous hemianopsia was readily demonstrated. The left pupil was larger; there was paresis of the left external rectus and extreme rotation to the left caused nystagmus. The pupillary reactions were repeatedly studied by Drs. Dercum and Gilpin. The left pupil was absolutely immobile to light, the right pupil failed to respond so long as the beam of light was thrown on the blind half of the retina; it responded by contraction at once when the beam of light was thrown on the side in which vision was preserved. A small pocket-light throwing a narrow beam was used. After about ten days the response became markedly less and finally disappeared so that the right pupil, like the left, became fixed to light.

The tumor was probably so situated as to give rise in a moderate degree to cerebellar symptoms and at the same time so far forward as to involve the optic tracts in relation with the primary optic centers or possibly these centers themselves. Dr. Dercum has seen the Wernicke sign four times and in one instance the clinical report was accompanied by necropsy findings. (See "Tumor of the Thalamus, More Especially of the Pulvinar, Presenting Wernicke's Pupil Reaction," *J. Nerv. & Ment. Dis.*, August, 1890.)

DISCUSSION

Dr. Charles K. Mills believed that this sign was important and capable of demonstration, although Philadelphia and New York ophthalmologists have denied the possibility of demonstrating it.

Dr. Mills was struck with the similarity between the case reported by Drs. Gilpin and Dercum and the first of several cases included in the paper of Dr. Seguin in the *Journal of Nervous and Mental Diseases*, volume 14, November and December, 1887, pp. 721-737, entitled "A Second Clinical Study of Hemianopsia; Cases of Chiasm-Lesion; Demonstration of Hemiopic Pupillary Inaction." In the case presented, the most important features are that the patient has almost entirely lost sight in the left eye and in the right eye has considerable impairment. This loss and impairment are apparently due to atrophy consecutive to a papilledema. The other symptoms point to a cerebral growth, and it is altogether probable that this neoplasm is located so as to involve the interior aspects of the right temporal lobe and to produce compression on the optic nerve between the chiasm and the pregeniculum.

In Seguin's first case there was loss of vision in the left eye, temporal hemianopsia with the right eye, complete atrophy of the left optic nerve, and partial atrophy of the right, with no other symptoms of cerebral lesion, and a demonstrable hemiopic pupillary inaction from examination of the right eye.

Dr. Mills believed it impossible to demonstrate the hemiopic pupillary inaction in the case presented as the methods adopted were not correct. It was of no avail to flash the light of an electrical ophthalmoscope to the right and left without closely restricting the illumination to one-half or one portion of each side of the retina. In order that this sign may be demonstrated it is absolutely necessary to take certain precautions and carry out a certain method.

He had on several occasions had this Wernicke pupillary inaction demonstrated for him by Dr. S. D. Risley and years ago he had been able with great difficulty to demonstrate the sign himself.

Dr. Langdon said that the best instrument at present was an instrument used for the transillumination of retinal detachments. It was a complete metal globe containing an electric light bulb, the globe having an opening on one side which was very small, and which projected a narrow beam of light; by the use of this the light could be thrown on any part of the retina desired, and then the only possibility of other parts being illuminated was by diffusion, which it seemed was impossible to be sure was overcome. At the University Hospital this has been tried on many cases of hemianopsia but without eliciting Wernicke's sign. Many ophthalmologists are skeptical concerning the possibility of showing the presence of this sign.

Dr. Gilpin said that the fact that this had gone on for four months, that he had such a marked choked disk, that he had some difficulty in coordination which showed more markedly than it did at the present time, led him to suspect that the lesion was within the cerebellar fossa and by interfering with the flow of the cerebrospinal fluid had filled up the lateral ventricle. He believed in some way pressure had been relieved so as to allow freer circulation. The boy was better than when he came in.

Dr. Gilpin said he knew that ophthalmologists take no stock in Wernicke's sign, but he was sure that in this case they had obtained it.

A CASE OF EXTRAPYRAMIDAL LESION. Presented by Dr. A. E. Taft.

E. Y., aged 9, had an unimportant family and personal history. She complained of weakness of the left side. Her left foot had always been weak but more so of late. During the past few months she had not used her left upper limb.

Examination.—There was some weakness of the lower part of the left side of the face. She showed her teeth with good bilateral innervation, but on elevating the corners of her mouth separately, it was done less well on the left than on the right side. The pupils were normal, and the tongue was protruded normally. The left upper limb was slightly smaller than the right. It was spastic and there was increase of tonicity both on active and passive movement. This was not constant in degree. Spasm of the limb prevented eliciting the tendon reflexes on this side. Those on the right side were normal. There were transient involuntary movements of the fingers, and occasionally of the forearms. These movements suggested beginning athetosis. The grasp of the left hand was good but weaker than the right. There was no disturbance of general sensation, stereognosis nor position sense. Lower Extremities: The left foot was turned in. The left lower limb was spastic; the right was normal. Tendon reflexes were somewhat exaggerated on the left, but not on the right. There was no sensory change, and no ankle clonus, but there was a Babinski reflex on the left. It is of special interest that the weakness of the left upper limb has been noted recently, while that of the lower limb was noticed when walking began.

DISCUSSION

Dr. Charles K. Mills said that the case was of great interest to him with regard to the existence, position and characteristics of the cerebral tonectic apparatus. This he believed had both a cortical and a lenticular representation. The cortical rendezvous of tone is probably in the midfrontal region. Dr. Mills noticed, in looking at this girl and also in testing her grip, that she probably had a species of preservation, although this was not marked. She had athetoid movements and did not seem to let go as quickly with the left hand as with the other. Therefore he thought that the diagnosis which had been intimated was correct, namely, an extrapyramidal, probably a premotor cortical or ganglionic subcortical, lesion.

A CASE OF BRAIN TUMOR OR ENCEPHALITIS ACCOMPANIED WITH DYSPITUITARISM AND SHOWING LONG CONTINUED PSEUDOSTUPOR. Presented by DR. CHARLES W. BURR.

Casual observation of the patient would give one the impression that he was unconscious; looking more closely the judgment would be apathy. He lay motionless, staring into space, paying no apparent attention to what was going on around him, responding only to painful stimuli. It was impossible to determine his real condition of consciousness because he was speechless, not from word deafness but because of mental dulness; he also had some defect of vision and was palsied on the left side. He was not in coma, but it is doubtful whether, when not irritated by external stimuli, he carried on any mental process or had any emotional tone. For want of a better name, Dr. Burr has called the condition of consciousness pseudostupor. At times when repeatedly spoken to he responded with a few words. He had been in this condition, not common in brain tumor, at least eight weeks. Dr. Burr had seen profound stupor, so deep that no stimulus caused evidence of consciousness, endure for four weeks in a patient with tumor of the left prefrontal lobe.

The patient was entirely too girlish in appearance for a boy of his years. He probably had some pituitary gland trouble, whether congenital or the result of inflammatory cerebral disease starting in 1918 is disputable.

The patient was 11 years old. The family and personal history was unimportant. The patient had had no serious illness until the onset of the present trouble in December, 1918, with a mild attack of influenza. Soon after he was up and about, his mother noticed that when he reached for an object with his left hand, his arm would fall slowly to his side. This condition grew worse and at the end of three weeks he was unable to lift the arm. He dragged the left leg in walking. At the beginning and for some time after, he complained of intermittent, aching pains, and numbness in the left arm and hand, left side of the chest and left leg. The fixed stare, so noticeable now, dated from December, 1918. This patient was seen by Dr. Spiller on Oct. 29, 1919, who made the following report:

Paralysis of upward and downward movements indicated the lesion to be in or near the corpora quadrigemina. There was no lateral ocular palsy. On looking to the right or left he looked down because of muscular weakness. He would probably have complete ophthalmoplegia finally. He could close the left eye tightly. The corneal reflex was normal. Masseter contraction was good. Weakness of facial muscles on the left side was present. The face was hyperesthetic. He had marked paresis of the left upper limb and marked spasticity of the left lower limb. Left patellar and ankle clonus was marked. There was great exaggeration of tendon reflexes, and a permanent Babinski reflex on the left with a slow Babinski on the right. Pain sensation was acute in both limbs; no loss of sense of position; stereognostic perception good; therefore no involvement of parietal lobe.

Summary: The lesion involved the right side of the brain above the anterior portion of the pons. It was very near and most probably just back of the corpora quadrigemina in the region of the basal ganglions and did not involve the cortex. The lesion was probably a glioma. There was probably no mechanical obstruction of the aqueduct of Sylvius causing internal hydrocephalus. The clinical picture has changed in many ways since Dr. Spiller made the examination.

A later examination by Dr. Burr showed that the pupils were fixed. He was unable to look upward with either eyeball. Horizontal movements were hesitant and jerky, and there were occasionally intermittent simultaneous nystagmoid oscillations of both eyes toward his nose. Rotary oscillation in the upward direction of the left eye was constant in the lateral position of the eyeball. Reflexes were normal on the right side, exaggerated on the left. The Babinski reflex was present on the left side only. Abdominal reflexes were absent. He was weak in the limbs on both sides, much more so on the left, and showed evidence of distinct hemiplegia. Speech was halting, stuttering and unintelligible. There was incontinence of urine and feces. A number of spinal punctures were made, the fluid coming out with great pressure. After one puncture the reflexes were reported almost normal. The cell count was 43 and there were no tubercle bacilli in the smear. The spinal fluid Wassermann test was negative. Roentgen-ray examinations showed the sella turcica to be unaffected, but there was internal hydrocephalus with separation of the sutures of the skull.

On Nov. 1, 1919, ophthalmologic examination showed that the disks were swollen and the veins tortuous; summit, + 7; fundus, + 5; the disks of the left eye were similar to those of the right but not so marked. Paralysis of upward movement and deficient convergence were present.

Physical Examination.—Examination when admitted to the Philadelphia General Hospital, Jan. 6, 1920, revealed a well nourished white boy. The skin was dry and soft and of rather delicate feminine texture. The contour of the extremities, as well as that of the trunk and pelvis was feminine. The genitalia were under-developed. The hair of the scalp was abundant. There was practically no body hair. There was no evident enlargement of the thyroid or other gland. The head and face were well developed. The patient was able to wrinkle the skin over the forehead equally on both sides. The masseters acted fairly well. His height was normal for his age. There was no mastoid swelling or tenderness.

The pupils measured about 3 mm. each; were fairly regular in outline and reacted sluggishly to light and accommodation. Extra-ocular movements could not be performed. He would not cooperate. He had a peculiar fixed, blank stare. He was able to close the eyelids.

The tongue protruded without tremor or deviation. Movements were imperfectly performed. There was no apparent difficulty in chewing or swallowing. The speech was somewhat thick, slow and inarticulate. (He talked little and only when strongly urged.) He was unable to expose the teeth on the left side properly.

The right upper extremity was freely movable in all directions. Biceps and triceps reflexes were preserved, but slightly active. Sensation to pinpoint and touch was preserved. On account of mental stupor the patient failed to recognize simple objects, such as a penknife and key, when placed in his hand. The left upper extremity was held flexed over the chest and somewhat rigid.

The chest was well developed. The mammae were too well developed and prominent for a boy. Thoracic organs were normal. Head Measurements: occipital protuberance to line of eyebrow, 10½ inches; circumference, 20¾ inches; meatus to meatus, 13¼ inches.

Eye Examination (by Dr. Langdon).—Pupils: 3 mm., the left was possibly a little larger; slight reaction even to concentrated light. The visual axes slightly diverged. Ocular rotations could not be studied. Vision: light perception only. The mediae were clear; disks very pale, especially the left; margins well defined, but with slight blurring; retinal veins about normal. The arteries were small, but without kinking or other changes. There were no other fundus changes; hyperopia 5 D; no cupping of disks.

In Blockley hospital his temperature has usually been 99; occasionally it has risen to 100, and three times, in the evening to 101. Blood examination, Jan. 14, 1920: Blood Wassermann test was negative. (Spinal fluid examination was not made because of possible brain tumor.) Blood culture was negative.

Roentgen-Ray Report, Jan. 14, 1920: There was apparently a thinning of the bones of the frontal region. Further than this no lesion could be demonstrated. Satisfactory stereoscopic plates of sella could not be obtained because the child moved during the examination.

Dr. Burr confessed that he was not sure as to the diagnosis. The boy either had a tumor—and if so, Dr. Burr agreed with Dr. Spiller as to the location—or he was suffering from encephalitis following, and probably the result of, influenza. Influenza was an exciting cause, the last straw, in many cerebral conditions. Dr. Burr said disturbance of consciousness, such as the boy's, was more common in encephalitis than in brain tumor. It was not coma, but mental sluggishness. Dr. Burr thought such a condition indicated a widespread affection of the cerebrum, just as trance in insanity indicates an affection of the whole intellectual cortex and not merely local disease.

A CASE OF CEREBELLAR LESION. Presented by Dr. Alfred Gordon.

J. O., a boy, aged 13, had been complaining of severe headache for the last two years. During the last six months he had had many attacks of vomiting without any indication of gastro-intestinal disorder. Vertigo began early and is still present but not continuous.

Examination.—When he turned his eyes sharply to the left, nystagmic movements appeared. Occasional and slight nystagmus was present when the eyes turned upward. Both nerve heads were hazy at the margins. There was some optic atrophy and the retinal veins were decidedly tortuous. The left temporal field of vision was diminished. Pain sense was diminished over the entire right side of the face. Hearing was impaired on the right. Reflexes, station and gait were normal. Pastpointing to the right was observed repeatedly in the right hand, but not in the left. A Bárány test performed by Dr. L. Fisher revealed: On turning the test to the right the duration was 38 seconds, to the left, 35 seconds. With the caloric test a douche on the right produced nystagmus of 50 seconds, on the left, of 70 seconds.

In view of the right pastpointing and the localization of the nystagmus, of the findings with the Bárány test, also of the eyegrounds, of the headache and vomiting, the presumption is in favor of a cerebellar lesion situated on the right side.

The ethmoids on both sides were badly diseased, and there was a profuse discharge of pus from both nares. The question arises as to the possibility of a purulent focus in the right cerebellar region. On the other hand, intracranial syphilis cannot be entirely eliminated in view of the fact that only the blood was examined for a Wassermann reaction, with negative results.

A CASE OF "PURE ALEXIA." Presented by Dr. Alfred Gordon.

A man, aged 72, right-handed, had emphysema of the lungs and endomyocarditis with arrhythmia; arteriosclerosis was pronounced. The blood pressure was: systolic, 190; diastolic, 120. The urine contained a small amount of albumin. The Wassermann test was negative and the eyegrounds normal. Several years ago he had had a mild attack of right hemiplegia from which he made a complete recovery. About one year ago he had an attack of vertigo with severe headache, following which he developed the present condition. He was unable to read printed or written words. Spontaneous speech, repeated speech, singing and understanding of spoken words were correct; reading aloud and mental reading were impossible. The patient was able at times to name some letters correctly, but was utterly unable to make out the sense of the word. Copying was difficult, but writing to dictation and spontaneous writing were almost entirely correct. However, the letters written were much larger than those of the patient's former writing. He could not read what he had written. He recognized an occasional word only when he followed each letter with his finger.

Two other interesting phenomena were present: First, verbal blindness concerned only words, not figures; the patient was able to add, subtract and carry out other arithmetical operations. Second, there was a right homonymous hemianopsia.

The clinical picture of the present case corresponds precisely to the description given by Déjerine in 1892 in which he isolated from Wernicke's sensory aphasia a form of aphasia to which he gave the name "pure word-blindness."

In alexia of the cortical origin due to a lesion of the angular gyrus, we usually observe an impairment of inner language; there is ordinarily a certain degree of word deafness, of paraphrasia and of agraphia for spontaneous writing and for writing on dictation. Moreover, not only words, but also figures are not recognizable by the patient. Finally a homonymous hemianopsia is rarely, if at all, observed in cortical lesions of the angular gyrus.

In the present case the lesion, which is a softening due originally to an embolic occlusion, is probably situated in the inferior longitudinal bundle, namely, fibers connecting the center of general vision with the center of visual images of speech (angular gyrus). Such a conception readily explains the absence of all other aphasic disorders and the presence of the hemianopsia.. We are dealing, therefore, with a case of "pure alexia."

THE CONDITION OF A PATIENT IN GOOD HEALTH, BUT EXHIBITING AUTOMATIC MOVEMENTS OF THE LOWER LIMBS, NINETEEN YEARS AFTER COMPLETE TRANSVERSE SECTION OF THE SPINAL CORD BY A GUNSHOT WOUND. Presented by Dr. Williams B. Cadwalader.

The case which Dr. Cadwalader described is the same one that Dr. Francis T. Stewart and Dr. Richard H. Harte reported in the *Philadelphia Medical Journal* of June 7, 1902, under the title of "A Case of Severed Spinal Cord in which Myelorrhaphy was followed by Partial Return of Function."

Their report caused much discussion at the time and the assertion that return of function had occurred in the lower limbs was received with a feeling of skepticism. Notwithstanding the fact that this patient was repeatedly examined by different physicians and was exhibited before medical societies, a definite conclusion was not reached.

Since 1902, such great advances have been made in our knowledge of the physiology of the spinal cord not only through the monumental work of Sherrington in the laboratory, but also by innumerable clinical investigations in man, and most particularly by those of Riddoch, Head and others on cases of complete division of the spinal cord from gunshot wounds observed during the war, that it seemed most important to record the present condition of Dr. Stewart's patient, nineteen years after the injury was inflicted. Dr. Cadwalader knew of no other patient who had survived so long after complete section of the spinal cord. It had been planned to make this report together with Dr. Stewart but he died suddenly three weeks before. Dr. Cadwalader said that Dr. Stewart had agreed with his views.

An abstract of the history as given in 1902 by Dr. Stewart and Dr. Harte follows:

C. N., 26 years old, was admitted to the Pennsylvania Hospital, Jan. 21, 1901, having been shot in the back with a 32 caliber pistol. The bullet had entered 1 inch to the right of the seventh dorsal spine and had passed directly into the spinal canal. There was immediate and complete motor and sensory paralysis below the level of the tenth dorsal spine and all the superficial and deep reflexes were abolished. Three hours after the injury, operation was performed by Dr. Stewart. The laminae of the seventh and eighth dorsal vertebrae were removed, the membranes were found lacerated and the spinal cord completely severed, the distance between the severed ends of the cord being three fourths of an inch. This observation was confirmed by Drs. Mitchell, Fraley and Newlin, who were present. The divided ends of the spinal cord were approximated with catgut sutures and the wound closed. Surgical recovery was uneventful.

After studying the history of this case, it appeared evident that the patient must have been in a state of "spinal shock" on admission to the hospital and that it lasted just three weeks, for it is stated further on in the report that the patellar tendon reflex was detected for the first time on the twenty-first day after the injury. Involuntary movements of the lower limb were noted on the thirty-fourth day. This must have been the beginning of the establishment of the condition called the "mass reflex." From then on the movements observed in the lower limbs could not have been voluntary movements, for at present it can be easily demonstrated that the patient is completely paralyzed in all portions below the costal margins and that the muscular movements formerly regarded as voluntary movements, are in reality the involuntary movements recognized as the "reflexes of defense" or the "automatic" or "reflex" movements that collectively make up what Riddoch has termed the "mass reflex" so essentially characteristic of complete division of the spinal cord.

Examination in 1919-1920.—There was complete paralysis of motion and sensation below the level of the tenth thoracic spine. The lower limbs felt rigid; there was no wasting. The skin over the legs was waxy in appearance and pitted slightly on pressure.

Involuntary flexor movements of the lower limbs occurred at irregular intervals. When sitting quietly, they might be absent for hours at a time, but on the slightest movement of the trunk or on cutaneous stimulation, marked flexor spasms occurred and if stimulation was rapidly repeated they became violent. The rigidity of the limbs was excessive, requiring considerable force on the part of the examiner to extend the leg but after complete extension had been accomplished, the limb was likely to resume gradually the partially flexed position. This was because of the predominence of the hypertonicity of the flexor muscles. When the patient was prone the limbs could be fully extended by the examiner and they would then remain so until some stimulus caused reflex flexor contraction.

With braces so arranged to extend up to and include part of the trunk and with circular metal strips about the lower leg to prevent complete flexion of the legs, the patient was able to stand erect between two chairs by first supporting herself with her arms and hands and then arranging her lower limbs in extensor spasm favored by gravity, and then removing the support of her arms, only using them to balance with. By this means she could stand erect in a purely "reflex manner" but only momentarily. Tendon reflexes could not be obtained because of rigidity. There was a bilateral Babinski sign, but much of the time the great toe was held in dorsal flexion as a fixed Babinski position. If the soles of the feet were strongly stimulated there followed upward movement of all the toes, dorsiflexion of the foot and strong flexion at the knee and hip.

On scratching the skin of the abdomen there was no muscular response.

Sensation: Sensation of all kinds was abolished. Nevertheless, she complained now and then of a feeling of fulness and discomfort in the abdomen, generally worse after excessive movements of the limbs, and she still had hallucinations as to the position of the toes or feet which were very annoying.

Bladder: The bladder emptied automatically. By practice and judgment she managed to anticipate evacuations and to get along fairly well, but she soils herself occasionally and sometimes wets the bed during sleep.

The patient's general health was good. She was most optimistic and believed that she had recovered voluntary motion. In making a sensory examination, her answers were most misleading. Sometimes when pricking her over the skin of the leg with a pin she seemed to answer correctly, though she did not feel the slightest sensation below the level of the lesion. By long practice she has learned to recognize the traction exerted on sound muscles of the thorax above the level of the lesion caused by the involuntary flexor spasm of the stimulated limb. If it were violent, she occasionally answered that she felt the pin. Moreover, she was so anxious to assist that she unconsciously deceived.

The transitory abdominal discomfort, i. e., a feeling of fulness and distress in the epigastrium and abdomen with nausea that she sometimes complained of, can be explained by assuming that it was the mechanical or reflex result of frequent flexor spasms of the lower limbs and traction on the abdominal muscles. It is significant that the patient stated that she could not vomit. Treatment for the relief of these sensations should be directed toward diminishing the violence and frequence of the involuntary movements by finding the best position for the paralyzed limbs and avoiding undue reflex stimuli. The patient had learned this by experience and fully appreciated its importance. She was cheerful and spent her time sitting comfortably and fully dressed in a wheel chair knitting sweaters, by which means she earned a good income. It must be denied emphatically that return of function had occurred; such a statement was entirely the result of a mistake in observation.

However, the case proves that complete division of the cord at the tenth thoracic segment by gunshot wound is not in itself necessarily fatal, for if the automatic bladder can be established, a patient can live a comfortable chair life indefinitely. Furthermore, it proves that even under the most favorable condition for surgical procedure—for the separated ends of the severed spinal cord were approximated by sutures only three hours after the injury—and after careful nursing for nineteen years, no clinical sign of return of function has appeared; therefore, it can be assumed that regeneration of the spinal cord does not occur, after complete section, and approximation of the ends by suture.

DISCUSSION

Dr. Andrew H. Woods said that the anomalous part of the case as reported was the spasticity in extension in a patient with complete severance of the spinal cord. In the cases reported by Sherrington, Head, Riddoch and others, when there was serious damage to the lateral columns or complete division of the spinal cord, there resulted frequent reflex spasms in flexion defensive in purpose; but there was not the tonic spasticity in extension, which appears to be nature's effort to prevent the body from falling when external disturbing forces have been encountered. The reflex mechanism through which this conservative stiffening of the lower limbs was effected apparently includes an afferent arc which extends from the muscle through the lateral columns of the spinal cord to the region of the midbrain; while the efferent arc according to Walshe possibly runs somewhere near the crossed pyramidal tracts. Hence any serious lesion in the lateral columns of the spinal cord would interrupt this reflex pathway. Dr. Woods thought the explanation given by Dr. Cadwalader of the patient's responses to unfelt sensory stimuli was ingenious and that it seemed adequate.

Dr. Alfred Gordon said that the toe phenomenon in a case of complete section of the cord deserves discussion. The opinion of the majority seemed to be that the toe phenomenon was a cutaneous reflex, dependent on the integrity of the cerebrospinal pathway.

Dr. Cadwalader said that it was true that in cases of complete division of the cord the paraplegia was flexor in type. In this case the spasm of the flexor muscles predominated, but the extensor muscles also showed definite hypertonicity at times. In this respect it did seem to differ from those cases described by Riddoch, Walsh and some other observers, but in spite of this Dr. Cadwalader thought because of the constant predominence of the flexor type of spasm, it should be regarded essentially as a paraplegia in flexion. The "mass reflex" is not so violent or active now as it had been formerly, and he believed this was in part due to the time that had elapsed since the injury, now nineteen years, and in part due to the fact that the patient had learned by experience to avoid all stimuli which provoked reflex action. In reply to Dr. Gordon's question, Dr. Cadwalader said that if the sole of the foot was lightly irritated the big toe would go up in a Babinski attitude, but if the sole of the foot was strongly irritated the toe and whole foot and leg were drawn upward in flexor spasm and would remain in this position some time, but gradually the flexor spasm relaxed and the limbs would lie almost fully extended on the bed. Dr. Cadwalader did not believe that partial preservation of the pyramidal tracts was neessary for such a reflex response.

A SPECIMEN SHOWING EXTENSIVE SUBDURAL HEMORRHAGE.

Presented by **Dr. George Wilson.**

Dr. Wilson said that his reason for showing the specimen was twofold; first, the long period which elapsed between the accident which produced the subdural hemorrhage and the onset of symptoms; and second, the absence of localizing signs.

History.—The patient from whom the brain was removed was admitted to the Philadelphia General Hospital on Nov. 11, 1919, to the service of Dr. Spiller and died November 15. The man's wife gave the following history:

He was well until four days before admission. He complained of headache on reaching home on the evening of November 6; two days later the headache was so severe that he stayed in bed and called a doctor He was sent to the hospital because of unconsciousness on the morning of November 11. Two months before the patient had had a fainting spell and fell, striking his head on the street car track. Since then he had had an occasional headache which had never interfered with eating, sleeping or working. His wife said that he drank to excess, but as far as she could determine, she was sure that he did not use drugs nor had he ever contracted a venereal disease.

Physical Examination.—The patient was unconscious. The pupils were unequal, the right being larger and did not react to light or in convergence. It was impossible to test for movements of the cranial nerves. There was bilateral choked disk of moderate degree. The deep reflexes were about normal. There was a Babinski sign on the left. There was no rigidity of the neck, no evidence of a fractured skull, and no Kernig's sign. The pulse on admission was 62, regular and of normal tension. The urine, blood and spinal fluid were normal. At necropsy a large, organized, subdural clot was found covering the greater part of the right hemisphere. There was no fracture of the skull. A few years ago Dr. Spiller showed a similar specimen to this society. It is indeed remarkable that a hemorrhage of the size shown here could exist for two months without producing any symptoms outside of an occasional transitory headache.

ON THE OCCURRENCE OF BILATERAL SYMPATHETIC OPHTHALMOPLEGIA IN LETHARGIC ENCEPHALITIS. **Dr. Williams B. Cadwalader.**

Dr. Cadwalader thought observation of this symptom exceedingly important because when paralysis of the sympathetic fibers supplying the eyes occurs on both sides it can only be caused by an inflammatory lesion situated in the upper cervical cord, medulla oblongata, pons or perhaps in the cerebral peduncles. The course and situation of the sympathetic fibers to the eye are well known in these portions of the brain, and it is in these parts of the brain that the inflammation is most intense in this disease. Therefore, when associated with the characteristic mental confusion bilateral paralysis of the sympathetic fibers of the eye should be regarded as a diagnostic sign of lethargic encephalitis.

In two cases Dr. Cadwalader said it had occurred early in the course of the disease, but because of the bilaterality it had not been recognized until other signs had developed. Narrowing of the palpebral fissure, myosis and slight retraction of the eyeball occurring on both sides can be readily overlooked whereas if they occur on only one side, they quickly attract attention. The smallness of the pupils in one of Dr. Cadwalader's patients had led to the suspicion of an overdose of morphia.

Recently a case was observed in the University Hospital in which the diagnosis was proved by necropsy and in one of Dr. Cadwalader's cases a postmortem examination had verified the clinical diagnosis.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Monthly Meeting, March 18, 1920

George A. Waterman, *President Pro Tem.*

DISSOLUTION OF CONTINUITY OF THE SPINAL CORD BY A PROJECTILE PASSING NEAR BUT NOT DAMAGING THE VERTEBRAL COLUMN. Dr. H. C. Solomon.

Dr. Solomon reported the case of a soldier, 22 years of age, wounded in battle, Nov. 3, 1918. The bullet wound was at the level of the eleventh dorsal vertebra, about a centimeter to the left of the median line. The next day roentgen-ray examination showed a foreign body in the right chest, said to move with respiration. He arrived at a base hospital a week later, at which time he had the symptoms of complete transverse myelitis: paraplegia, incontinence, etc. At this time he already showed several areas of decubitus—over the sacrum and both great toes. He lived until January 10, a little over two months after his injury. During this time there was no change in the paraplegic symptoms, but the decubiti increased.

Necropsy examination on Jan. 11, 1919, showed the body of a markedly emaciated white man, presenting a tremendous bed sore about 7 by 9 inches, extending from the left iliac spine to the right and from the level of the third lumbar vertebra to the anus, with the iliac spines visible and protruding. A small, superficial decubitus was present at the tuberosity of the ischium and decubiti in both popliteal spaces exposed the tibiae and communicated with the joints. There were also pressure sores over the great toes and right heel. Edema was present to the level of the waist. There was a small healed scar at the level of the eleventh rib behind, one-half inch to the left of the spinal column, undoubtedly the point of entrance of the bullet. The bullet track could be traced through the muscles in the lower dorsal region of the back. As far as could be determined, there was no injury to the bones of the spinal column. The foreign body, however, traveled in close proximity to the vertebral column, apparently passing between the transverse processes of the vertebrae. In the middle lobe of the right lung near its hilus, a piece of shrapnel approximately ¼ by ⅛ by ⅛ inch in size was found. The foreign body was walled off with no evidence of suppuration about it.

On removing the dorsal portion of the vertebral column, the dural sheath appeared to be entirely normal. On opening the dura, however, the cord in the dorsal region opposite the bullet track was found to be confluent, consisting of a grayish, liquefied mass. This condition was sharply localized, having a length of 1¼ inches. There was a small portion of the cord posteriorly and laterally that was not entirely liquefied. The pia above and below the injured portion of the cord was discolored a brownish black for an inch or so, apparently an old hemorrhage. The cord above and below this area had its normal shape and consistency. The brain and its envelopes and venous sinuses were grossly normal.

The other positive necropsy findings were: slight pericardial, pleural adhesions, discrete bronchopneumonia of the left lower lobe; slight dilatation of the ureters; cellulitis of the scrotum; acute cloudy swelling of the heart muscle, liver and kidneys and septic degeneration of the spleen.

The cause of the destruction of the spinal cord is not easily seen. Possibly the velocity of the bullet traveling in a closed space may have produced the damage by commotion. A somewhat similar case was reported by Froment (personal communication). In his case the paraplegia had been produced by a bullet passing in close proximity to the spinal column. The paraplegia improved after four or five months. The roentgen-ray findings showed no evidence of fracture.

DISCUSSION

Dr. Henry Viets: In Frazier's book on "Surgery of the Spinal Cord" there is a beautiful illustration of a French case in which the bullet passed near the vertebrae, cracked one of the laminae of the vertebra and caused a complete paraplegia. On necropsy examination no damage to the inside of the dura was found but the cord was completely cut across as sharply as with a knife. This is the only reported case that I have seen.

Dr. W. J. Mixter: I had a rather similar case. A boy was shot with a 22 caliber rifle. He had complete paraplegia. The dura was just nicked by the bullet which did not touch the cord. The cord showed fusiform swelling and its central portion was simply squeezed out. There was practically complete destruction of the cord except for a mere shell.

THE COURSE OF RECOVERY IN SPINAL CORD INJURIES. Presented by Dr. Stanley Cobb.

Dr. Cobb showed a chart on which were plotted the recovery curves of twenty cases of spinal cord injury. These cases were taken as the patients were admitted to Colonel Frazier's surgical service at U. S. Army General Hospital No. 11 at Cape May, N. J. They were studied for from three to nine months. The chart was made by dividing the abscissor into periods of thirty days and plotting against this ordinates in terms of the patient's ability or disability; in other words the heading of the lowest ordinate is "complete quadriplegia"; above this is "paraplegia" and then in order the headings "stands with support," "steps with support," "walks across the room," "walks a mile with support," and "walks a mile"; above this, "well." In this way the general symptomatology is plotted against the number of days since injury and a curve of recovery shown.

The chart showed three main groups of cases: The first group consisted of those whose injury was slight and was discovered only on routine examination. There were three such patients, all of whom showed no disability due to spinal injury at the time of their discharge. The second group was composed of twelve cases and was the most interesting part of the chart. These men showed complete disability after their injury for a period of from fourteen to forty days. They then showed some definite return of function and thereafter rapidly improved for a period of from three to five months. At the end of this second period their recovery curves flattened out and the condition remained practically stationary. The chart, therefore, indicates that in the initial stage of complete paralysis no accurate prognosis can be made, but if no return of function is shown at the end of forty days the prognosis is probably bad. If function begins to return at this time it will proceed for three or four months, after which there will probably be no more improvement. The third group was composed of five cases which showed no improvement at all from the time of their injury up to the end of the ninth month.

It was attempted to study the bladder, rectal and sexual symptoms in a similar manner, but they were found to be too irregular to chart. It appeared, however, that cervical injuries showed symptoms of this sort only when they were severe. The injuries in the lumbar and sacral regions almost invariably showed these symptoms but in an irregular way; for example, sexual power could be entirely lost and urinary control hardly injured at all, or vice versa.

DISCUSSION

Dr. Morton Prince: Some months ago I expressed skepticism regarding the claimed good results of operation. I wish now to point out the bearing of Dr. Cobb's chart on that statement. If an operation had been performed on Dr. Cobb's patients what paeans would have been sung here in praise of operation. I think we can't keep in mind too strongly the tendency to improvement without surgical interference in a certain number of apparently hopeless cases, as is shown by the history of the cases collected by Dr. Cobb and reported in his paper tonight.

Dr. W. J. Mixter: In regard to a question by Dr. Cobb as to whether surgery should come early or late, it seems to me that that question must be passed along by the surgeon to the neuropathologist to get his opinion as to whether the injury is operable if done early or whether compression of the cord can be relieved successfully at a late date. Personally, my late cases have been discouraging. The early cases have not been altogether successful either.

THREE TYPES OF SPINAL CORD INJURIES IN WARFARE. Dr. Henry Viets.

This paper will appear in an early issue of the Archives.

DISCUSSION

Dr. W. J. Mixter: I wish to add a word to what Dr. Viets has said about the difficulties of handling such cases in war work, particularly in handling them in a casualty clearing station. In a casualty clearing station under normal conditions with a certain specified number of wounded passing through, such cases can be handled fairly well. When the press of work comes, all the fine neurologic work may break down entirely. An examination may be made in a given case in order that a decision may be reached at once, but it is impossible to pass along the information thus gained to the next man. For that reason it is practical to hold these patients as nontransportables. Hospitals for nontransportables should not be placed too close to the lines on account of shelling and since during an advance everything may be lost.

Coming naturally into this discussion is the case of a woman at present at the Massachusetts General Hospital, who was shot in the back of the neck and the abdomen. The abdominal wound was cleaned up first as was natural. The wound in the back of the neck was explored far enough to find that the second cervical vertebra was cracked but the bullet was not found. It lies in the vicinity of the second vertebra, probably just inside the canal. She has at present a paralysis of the left leg which is slight, sensory paralysis of the right leg and almost perfect sensation of the right arm. She is in good physical condition. Her temperature and pulse are both 100. The question comes up as to what we are to do next. Are we going to try to extract the bullet or should we let it alone? The roentgen ray shows the bullet just over or just within the laminae of the second cervical vertebra.

Dr. Henry Viets: I have seen this patient of Dr. Mixter's one or twice. I think the important point is whether or not the bullet carried with it infection to the point where it now lies. If there is infection, as happened in most of the war cases, I think laminectomy is advisable. If the wound is not infected, it is a question whether to operate or not.

Dr. Stanley Cobb: In connection with the question of the noncatheterization of these patients, I think experimental evidence is of interest. I did a midthoracic cord transection on a dog. I left the dog alone hoping that in three or four days I would have an automatic bladder. I waited in vain. On the fifth day he died and necropsy showed an enormously distended bladder which filled almost the entire abdominal cavity. The bladder was so stretched that there were small hemorrhages all through it. I suppose the abnormal distention of the bladder may have paralyzed the sympathetic nerve. Other investigators who work on the spinal cord have told me that their animals die, as mine did, unless they are catheterized.

Dr. Henry Viets (in closing): Apparently the automatic bladder is of great use to the patients as months go by, but the point is that the infection is much more important than the automatic bladder. As I have tried to point out, urology should stand first and neurology second. I believe that noncatheterization is bad neurology, but if it will save the infection, it is certainly worth while.

CHICAGO NEUROLOGICAL SOCIETY

Regular Monthly Meeting, March 25, 1920.

G. B. Hassin, M.D., *Vice President, Presiding*

CASES WITH INVOLUNTARY MOVEMENTS FOLLOWING ENCEPHALITIS. Dr. Ralph C. Hamill.

This article is published in full in this issue.

A CASE OF LETHARGIC ENCEPHALITIS. Dr. Sigmund Krumholz.

The patient was admitted to Cook County Hospital, Feb. 13, 1920. He was 22 years of age, married, had good habits and no history of previous disease.

In the early part of October, 1919, he suddenly developed intense headache, vertigo, impairment of vision with diplopia. The headache persisted for some time and was replaced by lassitude, which gradually deepened into lethargy. In December left facial paralysis appeared, soon followed by weakness of the left lower extremity, and several weeks later by paralysis of the left arm. In January, epileptiform seizures set in, of which he had had eight by February 10. For about four weeks prior to coming to the hospital he had at times had difficulty in swallowing; choking and regurgitation of liquids. During all this time the patient was up and about until two days before admission, and he did not realize the gravity of his disease.

Examination showed a well-built, husky adult, in a stuporous state, with brief lucid intervals; the neck somewhat retracted, absence of facial expression, mouth puckered; response to questions with single words or nods of the head; with limited lateral motion of the eyes, and distinct external squint of the right eye. The pupils were unequal; they did not react to light and little in accommodation. There was spastic paralysis of the left side of the body and constant coordinate movements of the right arm and leg. The reflexes of all extremities were exaggerated but more so on the right. Babinski, Chaddock, Oppenheim and Gordon reflexes were positive on the left, negative on the right; the abdominal reflex was present. Kernig's sign was positive, Budzinsky negative; the temperature was 100.4 F., pulse 112.

Subsequent examination disclosed neuroretinitis; blood test, 4,380,000 red cells and 7,800 white cells; the differential count was normal; the Wassermann reaction negative. The urine contained a trace of albumin; no casts. The spinal fluid test showed normal pressure, clear color, positive globulin test, 15 cells; Wassermann and Lange reactions negative. Smears revealed no micro-organisms, and cultures were negative. Roentgen-ray examination of the skull was negative. Later the spinal fluid showed 8 cells.

The general condition of the patient fluctuated; the rigidity of the neck disappeared; there was transient, bilateral, external strabismus; the temperature ranged between 100.4 and 102 F.; the pulse between 100 and 144.

Diagnosis: Acute and subacute meningitis as well as cerebral syphilis were ruled out chiefly because of the spinal fluid findings. Brain tumor was considered, but the presence of neuroretinitis, no choking of disks, atypical development of paralysis, and especially the constant presence of fever and rapid pulse, inclined him to a diagnosis of encephalitis.

Note: The patient has died and necropsy examination revealed a tumor in the region of the third ventricle, which probably explains the presence of fever.

CASE OF ENCEPHALITIS WITH UNUSUAL SYMPTOMS. Dr. Julius Grinker.

A woman, aged 21, married, with negative family history and unimportant personal history had a severe attack of influenza a year ago.

On the evening of Feb. 13, 1920, she complained of slight headache in the right frontal region. The next morning she awoke apparently well. Two or three hours later she vomited, became cyanotic, developed stertorous breathing with weak, irregular and rapid pulse. When put to bed, it was noted that the pupils did not respond to light and that there was convergent strabismus. The neck muscles were stiff and there was complete left hemiplegia. At this time the tendon reflexes were absent on the left and present on the right.

The next day the hemiplegia continued and the patient appeared drowsy and apathetic. She answered questions intelligently when urged to respond. To use Foster Kennedy's phrase, she was "intellectually keen, but emotionally stupid." This mental state lasted about four weeks; gradually she began to take an interest in her environment and became very much awake.

The ocular findings were transient diplopia and strabismus. Even six weeks after the onset there was occasional paresis of the left sixth nerve.

White counts were made as follows: February 14, 24,950; February 15, 15,250; February 18, 9,350; February 23, 16,550; March 8, 11,450; March 12, 8,950; March 16, 7,050.

The Wassermann test of the blood and spinal fluid was negative. The spinal fluid was under tension and somewhat blood-tinged, but contained no micro-organisms.

The maximum temperature was 101 F. Since March 2 she had had no fever. The pulse had fluctuated between 95 and 120.

On the third day of the disease the optic disks were hyperemic. One week from the onset, there was definite optic neuritis, intense within the next few days and persistent up to the present. During the past week the appearance of the disk had changed somewhat; there were to be seen whitish areas denoting scar formation and there was better definition. One could readily discern improvement from day to day.

Another unusual symptom, observed only in the polio-encephalomyelitic type of the disease and not classed under the lethargic type, was the development of muscular atrophy in the face and upper extremity. About ten days after the beginning of the disease, left-sided facial peripheral paralysis was noticed involving all the branches of the nerve. The peripheral palsy was, so to speak, engrafted on the central facial palsy.

In addition to the peripheral or nuclear palsy of the left half of the face, there were noted within a few days a certain flabbiness of the muscles of the left upper extremity, instead of the expected spasticity. Two weeks after the onset, wasting of the forearm and hand muscles on the left was pronounced and was more marked in the extensors. The biceps was somewhat wasted and felt flabby; the triceps was only slightly affected. The palm of the left hand was atrophied. The left thumb and thenar eminence were atrophic. Sensation was normal. The triceps reflex was somewhat reduced on the left, the wrist reflex completely lost.

Improvement in the paralyzed muscles was rapid. The patient could now raise the leg from the bed. The upper extremity at first improved but soon the peripheral paralysis abolished all motion. At present the arm is again improving.

The left peripheral facial palsy was still present but greatly improved and there was diplopia when the patient looked to the left. The headache, troublesome at first and almost constantly present on the right side, was still in evidence at times but the patient was happy, took an interest in everything about her and begged to be taken home.

Conclusion: The case reported partakes of the symptoms of lethargic encephalitis, hemorrhagic encephalitis, polio-encephalomyelitis, cerebrospinal syphilis and brain tumor in which hemorrhage has occurred. The Wassermann tests on blood and spinal fluid were negative and clinical signs of syphilis were absent, making that diagnosis improbable. A radiogram for evidences of brain tumor, with special emphasis on the sella and the posterior fossa, gave no clue to such diagnosis. There remained for practical consideration only epidemic polioencephalomyelitis and epidemic encephalitis of the lethargic variety. Though lethargy was a pronounced symptom in this case, we were not dealing with an encephalitis only, but also with a poliomyelitis; in other words, a lethargic polio-encephalomyelitis.

A CASE OF THIRD VENTRICLE TUMOR MISTAKEN FOR LETHARGIC ENCEPHALITIS. Dr. Peter Bassoe.

A woman, 53 years old, was admitted to the Presbyterian Hospital on Jan. 14, 1920, Three or four months previously she became irritable, abrupt and seclusive, and lacking in self-confidence. On December 31 she failed to prepare supper for her husband, insisting that the meal had already been eaten.

The next day she complained of frontal headache; slept poorly for a few nights and rapidly became more confused. On January 7 she became bedridden and thereafter did not recognize people. Her conversation was always incoherent. She did not complain of headache except on the date mentioned. When examined on January 15 she was semistuporous and muttered incoherent words. She was rigid and constantly clenched the fists. The pupils and disks were normal. The facial expression was rather tense. No paralysis could be discovered. The knee jerks were weak; the right ankle jerk was present; the left absent. Abdominal and arm reflexes were not obtained.

Hemoglobin was 75 per cent., erythrocytes 4,600,000; leukocytes 13,400.

Spinal Fluid: The Wassermann test was negative; the Ross-Jones globulin test positive; cell count 98, mostly lymphocytes; Lange gold test: 0112332100.

Gradually she became more stuporous and more rigid and perspired freely. After a few days a Babinski sign was obtained on the left side and later on both sides. January 19 it was noted that the right eye was more widely opened than the left. The condition of the tendon reflexes varied; at times none was obtained. February 10 moderate left ptosis was recorded. Although very stuporous she could be made to eat quite well. Continued fever was present from the date of admittance, the temperature varying from 100 to 101 F. during the first week; after January 21 it ranged from 101 to 103 F. The pulse rate was variable, becoming rapid toward the end. A second lumbar puncture on February 5 yielded a clear fluid with a cell count of 5, weakly positive globulin test, and a weaker Lange reaction (0012221100). The patient died on February 20. The clinical diagnosis was lethargic encephalitis, and no other condition was seriously considered. It is regrettable that the eyegrounds were examined on only one occasion.

Necropsy Findings: The brain weighed 1,507 gm. after three weeks of hardening in formaldehyd. The pial veins were moderately distended, the convolutions broad and flattened, giving an external appearance of edema. There was some milky thickening along the superior longitudinal fissure.

On section the white matter in the frontal region showed decided hyperemia. Section across the anterior end of the lateral ventricles showed the left frontal horn to be markedly distended and the right one moderately so. A broad, grayish-red tumor mass formed a barrier between the two ventricles, being attached to the corpus callosum above and the orbital convolutions and the caudate nuclei below. It merged imperceptibly into the brain substance below, but was sharply defined from the corpus callosum. At this level the tumor measured 3.5 cm. from side to side and 2 cm. from above downward. A small cyst 2 mm. wide was seen in the tumor tissue.

A section through the infundibulum showed that the tumor filled the third ventricle and on the left side an oblong cyst 5 by 9 mm. projected into the left lateral ventricle; while a small sessile nodule projected into the right ventricle. The left foramen of Monroe was open, while the right one was slit-like and compressed by the tumor. The base of the tumor on the right side merged with the innermost portion of the basal ganglions and internal capsule. The tumor ended 1 cm. behind this level, the posterior part of the third ventricle being free. The aqueduct was considerably dilated. The lateral ventricles at this level were only slightly dilated, while farther back the occipital horns were much dilated. The most posterior part of the tumor contained a cyst which measured 12 by 8 mm.

DISCUSSION

Dr. G. B. Hassin thought the pathologic changes in a typical case of lethargic encephalitis were present in the cortex as well as in every other part of the central nervous system, affecting both parenchymatous and interstitial elements. The most striking parenchymatous changes show as neuronophagia, which is considered by some observers a rare or accidental phenomenon in encephalitis. In six cases studied by him, neurophagia was a constant finding. It was also present in Bassoe's atypical case of encephalitis and will be found in any other case, especially in the deeper cortical layers adjacent to the white brain substance, provided a sufficient amount of slides are studied. In an acute disease like lethargic encephalitis, lasting days or weeks, one cannot expect such widespread changes as in dementia paralytica or African sleeping sickness, which are chronic diseases of years' duration. Even in these types of encephalitis, many ganglion cells may be found in a practically normal condition. Absence of parenchymatous changes is rather inconsistent with the diagnosis of encephalitis. Their unusual localization or severity may be responsible for the unusual symptoms in cases like those of Bassoe, Hamill and possibly Grinker. Unfortunately, pathologic studies of such abnormal localizations are lacking, Dr. Bassoe's case being so far the only one more or less carefully studied. Pathologic studies are absolutely essential for establishing the true nature of many atypical cases reported and demonstrated as lethargic encephalitis, some that are decidedly another condition, for instance, a tumor.

Dr. Julius Grinker stated that a patient with lethargic encephalitis under his care at present had often made him think of brain tumor that might give similar symptoms, and if the patient went to exitus he would not be surprised to find the same things that Dr. Bassoe had described. At present the patient was making a splendid recovery. He thought the diagnosis should be made with great caution, but as a rule one need not wait for postmortem findings to diagnose lethargic encephalitis.

Dr. A. S. Hershfield asked Dr. Bassoe what the eye findings were in the case of tumor.

Dr. Bassoe replied that the eyegrounds were normal when the patient was admitted to the hospital, but they were not examined again.

Dr. Grinker asked whether any one had ever seen optic neuritis in poliomyelitis.

Dr. Hamill replied that he saw a case in a child 1½ years olds, who had typical poliomyelitis with choked disk. Lumbar puncture was done and the necropsy showed marked polio-encephalitis.

Dr. Grinker said that about three years ago he sent a woman of 30 into the Cook County Hospital in a state of infectious delirium. Examination revealed typical choked disk and slight atrophy of the hand muscles and marked foot drop on one side. The patient was blind. At that time Dr. Nuzum was making his first experiments with the serum treatment of poliomyelitis and he administered two doses to this patient. She made a perfect recovery and could see again. The case was a typical polio-encephalomyelitis with optic neuritis and it was his experience with that case which enabled him to reconcile the optic nerve findings with the diagnosis of lethargic polio-encephalomyelitis in the case just reported.

Dr. Hugh T. Patrick thought the most confusing thing about lethargic encephalitis was the multiplicity of types. Some of the cases began with intense headache, other patients did not have headache. Some, as Dr. Grinker said, presented quite a typical picture of toxic delirium, in others the condition was strongly indicative of meningitis, with marked retraction of the head, stiffness of the neck and a Kernig's sign, with rather clear sensorium. Another peculiar mental state was one of apparent lucidity with severely impaired circulation. He had seen one young man with marked retraction of the head, a distinct Kernig's sign and feeble reflexes, fever not high, who seemed quite clear mentally. He looked alert and answered questions as to how he felt, quickly and correctly, presence of pain, etc., but when asked where the pain was located he did not know. He did not know he was in a hospital, or what city he was in and could give no account at all of the last two weeks.

He had seen a similar case in an older man. His family said he was all right mentally, but questioning revealed that he was disoriented and badly confused about familiar things. This case was quite suggestive of rapidly progressive vascular trouble, especially as he had had several peculiar attacks of transient dizziness and confusion.

Then there was the type with rigidity and facies of the Parkinson type; these patients were generally quite rational and answered questions intelligently. At the time he saw one patient of this type an attorney was there to draw the will and it was obvious that the man was competent to make a will. He answered questions concerning finances and what he wished done with entire lucidity, and yet when he was let alone, one would say that he was probably semiconscious if not unconscious; but that was in appearance only.

Some of the cases looked much like cerebral or cerebrospinal syphilis, and some apparently had a marked functional element. Dr. Patrick had recently had under observation a woman who acted like an old hypochondriac or psychasthenic; she worried constantly about herself and repeatedly asked him whether he was sure she was not going to die. This patient had persistent tachycardia, with no apparent cause. Some patients complain of numbness, tingling and dryness of the mouth, others develop peculiar contractures and still others odd tremors or movements reminding one of athetosis or chorea.

Dr. Patrick asked Dr. Bassoe whether he had formulated anything on which he could base a prognosis, and confessed that personally he could do nothing but guess at the outcome. He cited two contrasting cases: A young man was decidedly disoriented, with marked Kernig's sign and strongly retracted head. He was apparently very ill and made a rapid recovery. He had been seen only two and a half months ago; since that time he had recovered, married, gone on a wedding trip, returned and was at work. At about the same time he saw a young woman in about the same condition, except that she had severe headache and was slightly delirious, though she could be roused and answered questions rationally. She died four or five days later. When seen her condition did not appear to be much more serious than that of the young man.

Then there were the patients who passed through the acute stage and went along with some disability. What was the ultimate outcome in these cases to be? Would they be left with some physical or mental impairment, or both?

Dr. Patrick also asked whether Dr. Bassoe had found any treatment that was of value.

Dr. Bassoe said he could not answer Dr. Patrick's question in regard to prognosis and treatment.

CHICAGO NEUROLOGICAL SOCIETY AND CHICAGO PATHOLOGICAL SOCIETY

Joint Meeting, April 12, 1920

Lewis J. Pollock, M.D., *President of the Neurological Society, in the Chair*

THE MORPHOLOGY OF THE SYNAPSE IN VERTEBRATES. George W. Bartelmez, Department of Anatomy, University of Chicago.

In no field of investigation has the personal factor played a more prominent part than in the study of the ultimate structure of the central nervous system, especially in the matter of the synapse. The chief reasons for this are the complexity of the tissue, the minute proportions of the structures involved with the attendant difficulties of interpreting the microscopic picture and also the prominence which technical procedures must play in the study. Since the neuron theory accepted the Cajal doctrine of the synapse by contact it has been most successfully attacked from this aspect. If one be inclined to question whether the Golgi and Cajal methods give complete pictures of the strictly nervous elements of the tissue, then the bulk of evidence at the present time favors protoplasmic continuity at the synapse. This is significant since the findings favoring continuity are positive as contrasted with the negative findings for contact. On the other hand, our present knowledge of the structure of protoplasm and of the effects of current histologic methods on it are beginning to imbue us with a hopeful skepticism concerning the reliability of most cytological preparations, particularly of the central nervous system. We need more perfect methods and there can be little doubt that, when developed, they will discredit much generally accepted work.

No other tissue approaches the central nervous in complexity, so it is worth while to enumerate the number of histologic elements which enter into the composition of the gray matter. Five groups of structures are recognized:

(*a*) The undoubtedly nervous elements: the perikarya, their dendrites, axons with collaterals and the ramifications of afferent axons.

(*b*) The neuroglial elements: the protoplasmic glia syncytium which is probably the Golgi net of Bethe, the neuroglia fibers and in most preparations the products of their disintegration.

(*c*) Myelin sheaths or the products of their partial solution such as neurokeratin nets.

(*d*) The fluid substratum or ground substance which certainly contains coagulable proteins.

(*e*) Blood vessels with their accompanying collagenic tissue.

All of these must be taken into account and besides there are the distortions following the solution of various elements and the shrinkage due to dehydration and embedding which add to the difficulty of interpreting the microscopic picture. It is essential that very thin sections be used if reliable observations are to be made and even then it is difficult to understand oblique sections.

Nowhere has the personal factor so insidiously entered into the problem as in the technical procedures. Since no one has thus far been able to study a living uninjured nerve cell or the effects of various fixing fluids on it, the criteria of a well preserved preparation have remained largely subjective. With the development of special methods for the various constituents of nervous tissue the tendency has grown to place implicit faith in the microscopic preparation. The study of many methods has convinced me that in most types of synapses the appearance of contact or continuity can be produced by using appropriate fixatives or by sufficiently hydrating the coagulated protoplasm with long washing, treatment with alkaline solutions, etc. Thus there can be no doubt that the descriptions of so careful an observer as Hans Held are perfectly objective, but I believe that his methods have produced fundamental changes in the relations of the nervous elements in the synapse. All who have worked largely with material fixed in formol and especially with the

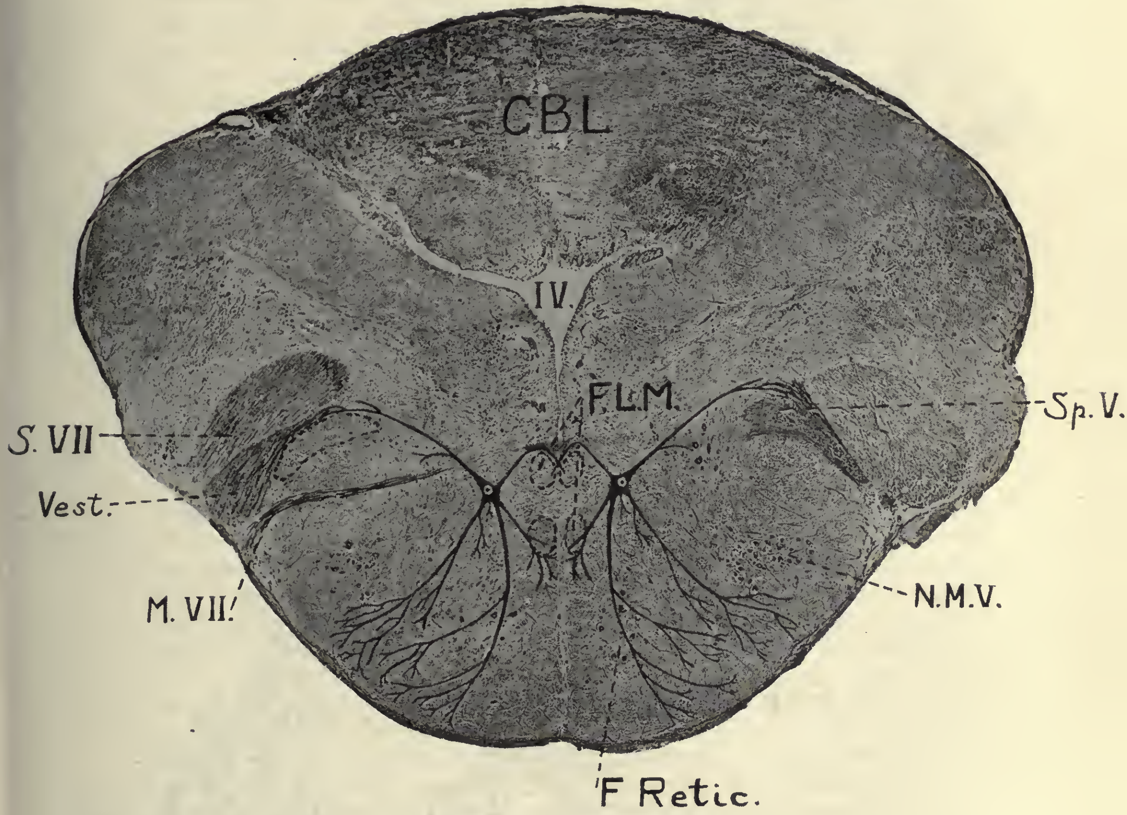


Fig. 1.—A photomicrograph of a section taken through the brain of a catfish at the level of the two Mauthner's cells. These cells have been reconstructed from the neighboring sections and projected into a single section. The ventral dendrites spread through the whole formatio reticularis. The lateral dendrite extends as far as the spinal fifth tract. It is surrounded by the fibers of the vestibular nerve, certain of which terminate on it in large club endings. The axons of the Mauthner's cells cross immediately and extend throughout the spinal cord in the fasiculus longitudinalis medialis. *CBL,* cerebellum; *F.L.M.,* fasiculus longitudinalis medialis; *F.Retic.,* formatio reticularis; *IV,* fourth ventricle; *M.VII,* motor root of N. facialis; *N.M.V.,* motor nucleus of N. trigeminus; *Sp.V,* spinal fifth tract; *S.VII,* sensory root of N. facialis; *Vest.,* vestibular root fibers.

Bielschowsky technic have supported the continuity theory, a fact which is to be associated with the swelling effect of the formol solution on nerve fibers even when it has been "neutralized" with magnesium carbonate. The only material in which nothing is stained but indubitable nervous structures is a successful Cajal preparation. Of course in the case of most synapses there is no answer to the criticism that the method does not stain *all* the nervous elements; but in at least one case there is an answer, as will appear shortly.

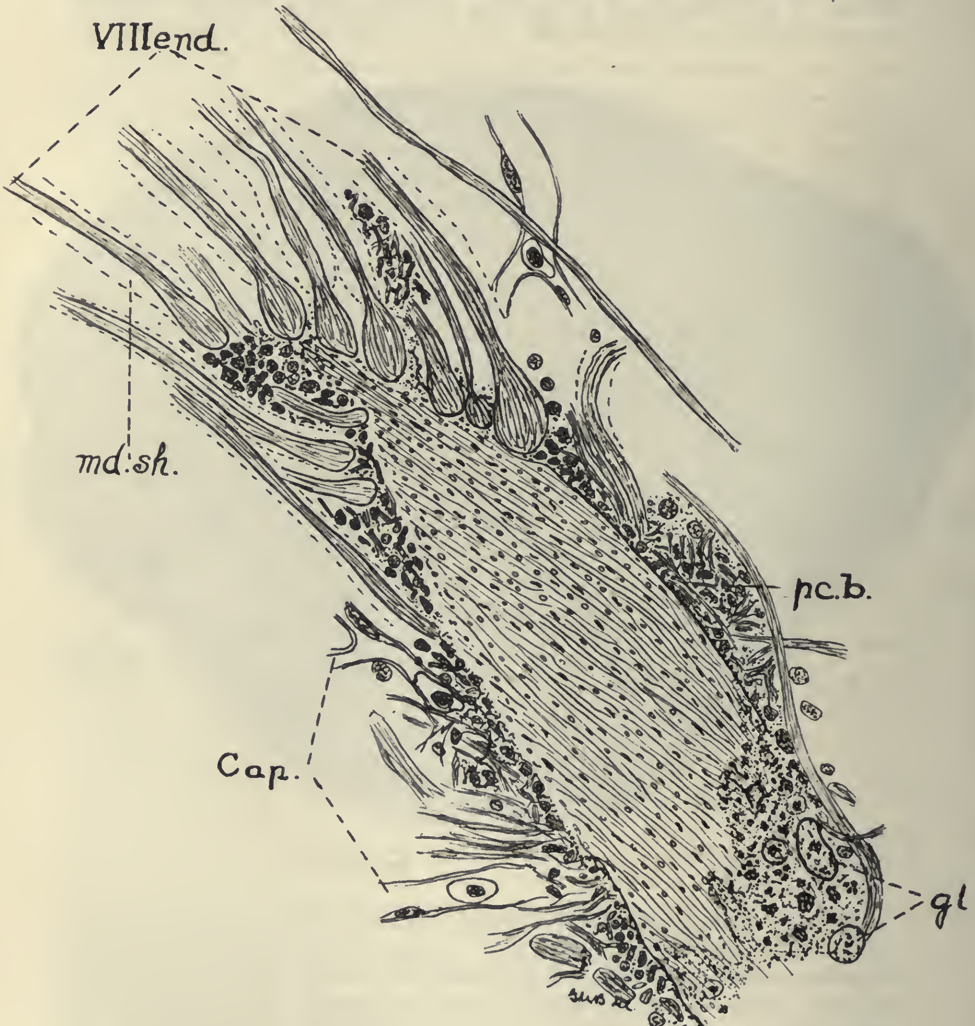


Fig. 2.—A portion of the lateral dendrite of the left Mauthner's cell of a catfish with the club endings of the thick vestibular root fibers ending on it; × 525. In the lower right hand corner of the figure the pericellular basket is shown in surface view where it spreads over the obliquely cut dendrite. Two glia nuclei show here at the edge of the dendrite. *Cap.*, capillaries; *gl.*, neuroglia nuclei; *md.sh.*, medullary sheath of a root fiber; *pc.b.*, pericellular basket; *VIII, end.*, vestibular root fibers with club endings.

Most other preparations show additional elements, thus complicating the picture and these are preferred by the advocates of continuity. Metallic impregnation methods come nearest to giving a specific stain for nervous elements but even here the specificity is only relative. Slight changes in the method may give very different pictures and when the glial elements are visible it is usually impossible to distinguish them from nervous elements by color alone; no matter how good the preparation, it is never possible so to differentiate them throughout an entire cross section of the brain. In any case it is essential that the silver methods be checked by other methods which have been shown to produce minimal change in form relations when used on tissues which can be studied fresh. Unfortunately the ubiquitous lipoids of the nervous system reduce many of the reliable fixing agents so rapidly that it is impossible to obtain adequate fixation even in small pieces of tissue. Unless the lipoids are at least partly preserved, the distortions due to their solution are so great as to invalidate any conclusions concerning the minute structure of most synapses.

Up to the present time only one synapse has been found in which the elements are large enough to give unambiguous microscopic pictures in all types of preparations. In 1915 I called attention to certain large bulbous endings of the vestibular nerve on the lateral dendrite of Mauthner's cell in the catfish, *Ameiurus melas* (*J. Comp. Neurol.* **25**:87, 1915). Here giant vestibular fibers terminate in club endings, the largest of which are 7 microns, the average being 4.6 microns in diameter as measured in sections. The endings lie in contact with a sword-shaped dendrite of a large cell, the axon of which extends throughout the spinal cord and makes direct connections with all the motor centers concerned in swimming. It readily shows the effects of adverse conditions as do other highly specialized cells in the nervous system. Marui (The Finer Structure of the Synapse of the Mauthner Cell, *J. Comp. Neurol.* **30**:127, 1919; ibid. **30**:253, 1919) has studied Mauthner's cell and some of its synapses under both normal and experimental conditions, but unfortunately he did not find these endings on the lateral dendrite which would have given him really clear evidence. This failure is probably due largely to a lack of familiarity with the teleost brain. Since he came to the conclusion that fibers from the dense pericellular feltwork enter into the body of Mauthner's cell, I have gone over the field again and come to the conclusions stated above; namely, that the technic plays a decisive rôle in the study of such synapses as Marui worked with. In thick sections (8 to 10 microns) of formol fixed material, especially in Bielschowsky and Levaditi preparations, one can find pictures which may be interpreted as evidence for the continuity theory of the synapse, but only in the very delicate synapses about the cell body. On the other hand, in every preparation I have studied, and this includes formol material, the histologic picture of the club endings on the lateral dendrite can be interpreted in only one way: viz., the vestibular endings in every case lie close to the lateral dendrite but never show any indication of fusion with it. This leads me to view with the utmost skepticism the reality of the conditions which have been observed by Held, Bielschowsky, Bethe, Marui and others in histologic preparations where there was continuity between different neurons (Heidenhain: Plasma und Zelle in Bardeleben, Handbuch der Anatomie des Menschen, **8**). That is to say, I believe that Held's pericellular nervous terminal net, the fusion of the protoplasm of "endfeet" with that of perikarya, the entrance of pericellular fibrils into the substance of nerve cells and similar conditions are postvital fixation or maceration artefacts. Experiments are in progress to test these conclusions in greater detail.

Marui (1919) has recently published the first study on the synapse under experimental conditions. In the much more complex synapses of Mauthner's cell which he studied, no changes in the purely nervous structures were found. His findings in the activities of the glia cells under experimental conditions in exhausted animals are of prime importance, but it remains to be seen whether these were due merely to the harsh method of "fatiguing" or to serious injury of nutritional origin.

DISCUSSION

Dr. C. Judson Herrick said Dr. Bartelmez was frank in expressing himself as a neuronist, with the preconceptions which this implies. He thought the majority of neurologists had been, or still are, neuronists. The facts in the case were clear in this particular instance. The histologic pictures, which he had personally examined, were as unambiguous as the histologic pictures one was in the habit of getting in studies on other tissues.

One thing must not be forgotten: Suppose we have a membrane which bounds the protoplasm of the end of the axon as it comes in from the vestibular nerve, and a second membrane which bounds the cell body to which this ending is applied, the nature of these membranes must not be neglected; they are not inert substances, but living tissues. It is still an open question whether to speak of protoplasmic continuity or not. The preparations appeared to show that there were two kinds of living substance intervening between the protoplasm of the first neuron and that of the second. That there was something there which was of tremendous physiologic importance, every one believed, and if we have a contrast between the protoplasm of the first neuron and that of the second and a further contrast between the two protoplasms that constituted the two membranes, there was some ground for the doctrine of discontinuity. Nevertheless, they were all living substances and so it was largely a question of definition whether to speak of protoplasmic continuity or not.

THE RELATION OF THE CEREBRAL HEMISPHERES TO ARTERIAL BLOOD PRESSURE AND BODY TEMPERATURE REGULATION.

F. T. Rogers.

This article will appear in an early issue of the Archives.

DISCUSSION

Dr. S. A. Matthews said that Isenschmid and Kiehl (*Arch. f. exper. Path. u. Pharmakol.* **70**:109, 1912) have published experimental results indicating that the caudate nuclei and the anterior part of the optic thalami may be recovered from rabbits with only a temporary change in the heat regulatory mechanism. When, however, the posterior and ventral portions of optic thalami were removed the animals became poikilothermal. Also Citron and Leschke (*Ztschr. f. exper. Path. u. Therap.* **26**:161, 1913) assert that destruction of the median portion of the between brain on the boundary of the corpus quadrigeminum and the optic thalami anterius makes rabbits poikilothermal.

In dogs it is quite easy to gain access to the third ventricle by drilling a hole about 5 mm. in diameter through the sphenoid bone just back of the optic chiasm. Of course, this will encroach somewhat on the pituitary body, but through such an opening lesions can be made in the basal ganglions.

In one dog in which he made a lesion in the anterior part of the optic thalami and which involved the principle, accessory and dorsal nuclei of the tuber cinereum, and after which the animal lived for three weeks, the temperature gradually fell and during the last two days of life was as low as 22 C. The animal gradually developed a lethargic condition.

When the animal was placed in an incubator at from 31 to 32 C. the temperature would go up to normal, and the reflexes would approach a normal state.

Evidently, from Dr. Rogers' results on birds and the results of others on mammals, the thalami contain the seat of the heat regulation. Just how they bring this about, whether by increased heat dissipation or by diminished oxidation has yet to be shown.

Dr. I. Leon Meyers asked whether any examinations of the temperature or arterial pressure had been made in dogs after producing in them the state of decerebrate rigidity.

Dr. Lewis J. Pollock asked what was done in these operations relative to closing the cranium so as to keep intact the intracranial pressure as this would probably have considerable to do with the general blood pressure.

Dr. F. T. Rogers (closing) said that the three questions are all significant. In reference to Dr. Meyers' question, this point has been tested out in the dog and it is known that the loss of the cerebral hemispheres leads to a depression of the blood pressure. In the dog there are certain complications however, due to anesthesia and to changes in the muscle activities involved in decerebrate rigidity.

Dr. Matthews has struck the keynote of the problem in referring to calorimetric controls in this work. It is evident that in the temperature regulation one must consider both heat production and heat loss. The heat loss in the bird seems to be principally through the skin and secondarily through the lungs by expiration. As to heat production, he was inclined to think, on the basis of evidence not yet conclusive, that there is deficient heat production resulting from diminished muscle activity.

As to Dr. Pollock's remark, his is the same question that has been raised by others in the last few years. In an article recently published by L. M. Moore, it is stated that a rise in body temperature does not follow puncture of the corpus striatum unless it is associated with increased intracranial pressure. In this report cases are not included in which there is clear evidence of increased intracranial pressure.

Furthermore, it may be added that it is an open question as to how much of the effects of hypophosectomy or irritation in the hypophoseal regions is due to secondary disturbances of the adjacent brain stem.

STUDIES ON THE SYMPATHETIC NERVOUS SYSTEM. S. Walter Ranson.

It is probable that the sympathetic nervous system is constructed along somewhat simpler lines than those indicated by the description found in most textbooks of histology. It is well established that visceral efferent fibers run through the ventral roots and white rami to the sympathetic trunk, within whose ganglions the majority of the fibers end. Some are continued through the splanchnic nerves to the ganglions of the celiac plexus. These fibers end in synaptic relation with neurons of a second order, often designated as sympathetic neurons, whose axons run to end on involuntary muscle or glandular tissue. Thus two visceral efferent neurons are concerned in transmitting an impulse from the cerebrospinal nervous system to smooth muscle or gland, and these are often designated as preganglionic and postganglionic with reference to the ganglion which contains the synapse between them.

There are probably no afferent neurons in the ganglions of the sympathetic trunk nor in the collateral ganglions, and no afferent fibers running from these ganglions to end in the spinal ganglions. Dogiel's work, published in 1896, in which he distinguishes both sensory and motor neurons, has not been supported by more recent investigations. Langley has shown, and we have been able to confirm his observations on this point, that after section of a spinal nerve distal to the spinal ganglions all or nearly all of the fibers in the corresponding white ramus degenerate, which would not be the case if any considerable number of afferent fibers of sympathetic origin were directed through the rami into the spinal ganglions. The afferent fibers of the sympathetic trunk and splanchnic nerves are probably all derived from cells in the spinal ganglions.

Sections of the superior cervical ganglion of man, stained by the pyridin silver technic, display a wealth of dendritic branches arising from the multipolar ganglion cells. These are of two kinds: namely, subcapsular dendrites and extracapsular dendrites. The latter pierce the membranous sheath or capsule, which surrounds the cell, and run long distances in the ganglions, branching repeatedly. The subcapsular dendrites branch and intertwine beneath the capsule, causing it to bulge locally and forming what may be called a glomerulus. When the localized subcapsular mass of dendritic ramifications is formed from the dendrites of one cell, it may be called a simple glomerulus. If formed by dendrites from two or more cells it may be designated as a bicellular, tricellular or multicellular glomerulus.

The extra capsular dendrites interlace with untold numbers of fine branching axons, forming an intercellular plexus which occupies the interspaces among the ganglion cells. We are interested chiefly in the axonic ramifications which help to constitute this plexus. Huber and Dogiel asserted that the majority of these fibers were the axons of sympathetic cells in the same or nearby ganglions; and their observations on the composition of this plexus constitutes the only histologic evidence worth mentioning for the existence of association neurons in the sympathetic system. Such neurons, if present, would serve to unite one ganglion with another or the several parts of one ganglion with each other. The physiologic evidence, so well presented by Langley, is all against the existence of such association neurons. The important question then is: What is the source of the fine axons in this plexus?

In a number of dogs we cut the cervical sympathetic trunk, thus severing all of the preganglionic fibers which run to the superior cervical ganglion. After time was allowed for degeneration, pyridin silver preparations were made of the superior cervical ganglion. These showed a complete absence of the fine branching axons of the intercellular plexus. These experiments furnish strong evidence that no synapses are present in this ganglion except those between preganglionic and postganglionic neurons.

The sympathetic trunk of the cat consists of more or less segmentally arranged ganglions bound together by fibers from the white rami. Preganglionic fibers from the upper nine white rami run upward in the trunk to the stellate and superior cervical ganglion. Similar fibers from the white rami below the fourth run downward in the trunk and many of these run through the splanchnic nerves to the celiac plexus. Others terminate in the ganglions of the thoracic, lumbar and sacral portions of the trunk.

The visceral afferent fibers of the sympathetic trunk and splanchnic nerves take origin from the cells of the spinal ganglions. They include large, small and medium-sized myelinated fibers and many which are unmyelinated. These were studied in white rami from which all the preganglionic fibers had disappeared as a result of section of the corresponding spinal nerves proximal to the spinal ganglions. Such partially degenerated rami containing only sensory fibers could be followed into the trunk by a study of serial sections. In the trunk each such ramus forms a longitudinal fascicle which maintains its identity for long distances, and the fascicles from the lower thoracic white rami can be traced into the splanchnic nerves.

The splanchnic nerves of the cat usually contain no appreciable number of postganglionic (i. e., sympathetic) fibers. These nerves are composed of preganglionic visceral efferent fibers from the spinal cord and visceral afferent fibers from the spinal ganglions, both of which reach the sympathetic trunk by way of the white rami and merely run through that trunk to enter the splanchnic nerves. In two of ten greater splanchnic nerves examined there were also present small bundles of postganglionic fibers. In one of these instances it was obvious that these fibers arose from an extra ganglion located on the sympathetic trunk between the first and second lumbar sympathetic ganglions.

These observations have been reported in greater detail in volume 29 of the *Journal of Comparative Neurology.*

DISCUSSION

Dr. C. Judson Herrick asked whether Dr. Ranson had extended his observations to the peripheral ganglions, and if he could give any suggestions as to whether they would apply equally to the ganglionic complexes of the abdominal viscera, etc.

Dr. Ranson (closing) thought Dr. Herrick's question was pertinent. He was sorry to say that he had not been able to get a satisfactory preparation of the peripheral ganglions. The technical difficulties involved in the study of the peripheral ganglions are much greater than those encountered in the study of other parts of the sympathetic system. He had tried to be careful to limit his remarks to the sympathetic chain, splanchnic nerves, and the prevertebral ganglions. In regard to these, the physiologic and histologic evidence is in full accord; but in the case of the peripheral ganglions, the physiologic evidence seems to point to something which we do not have in the ganglionic chain, a mechanism for visceral reflexes that is independent of the cerebrospinal axis.

THE NEW YORK NEUROLOGICAL SOCIETY

The Three Hundred and Eightieth Regular Meeting, April 6, 1920

Walter Timme, M.D., *President, in the Chair*

PRESENTATION OF CASES OF FAMILIAL DYSTONIA MUSCULORUM OF OPPENHEIM. Presented by Dr. Isadore Abrahamson.

Dr. Abrahamson presented three patients in one family, illustrating different stages of the disease. They were of the pure idiopathic variety, noteworthy especially, first, for their definite familial character; second, for their resemblance in the deviations from type that are to be encountered in all familial diseases; third, for a distinct involvement of speech, which Oppenheim denied in his cases; fourth, for the involvement of the musculature of the neck not common in these cases; fifth, for the varying mental attitude in the three patients, and sixth, for an unusual propulsive phenomenon which had not previously been noted in cases of this sort.

The first patient, a young Russian woman of 20, with unimportant family and personal history, at the age of 12 experienced difficulty in writing and became clumsy in her gait. The muscles of her legs would stiffen, the stiffening gradually spread to other muscles and was particularly increased by effort or emotion. While trying to lie quietly her muscles stiffened and caused her extreme annoyance. On attempting to grasp an object, a coarse tremor appeared. Her symptoms became aggravated during the last three months before presentation so that she could neither sit nor walk. During sleep all symptoms disappeared. The muscles were hypotonic. There was no paralysis. An abnormal wrinkling of the forehead and general anxiety were deviations from the usual Oppenheim manifestations. Speech was dysarthric, of bulbar type. The legs were paralytic, usually kept crossed, the typical inward rotation of the thigh was clearly apparent, and the feet were turned down. The Adam's apple was prominent, and she showed torticollis. Dystonia, tortipelvis, lordosis, clonic movements of the left hand with tonic movements in legs, and a rhythmic tremor characterized the disease in this patient.

In the second sister, aged 16, the disease had an insidious onset. She first noticed difficulty in writing, her muscles began to stiffen involuntarily, the left arm was drawn up in flexor spasm, the hand turned outward. She complained that the hip joint on the right would not stay in place, and a drawing feeling above the knee caused her to incline forward, producing a type of propulsion that is very interesting. Her body was thus bent forward and also laterally twisted around the vertical axis of the spine. There was inward rotation of the thigh. The gait was bounding. Her condition was much improved when feeling well and rested.

The third member of the family, a boy of 15, showed the disease in its early stages. His first difficulty was also with writing. He had the inward rotation of the thigh, the toes pointed down, the heel was carried high. This condition had slowly been getting worse for the last two years. In walking he swished his foot, and there was a slight forward bend. He ran well but had difficulty in stopping. He could make some hand movements such as threading a needle and playing marbles. A fine tremor was noted. Scoliosis and tortipelvis were present.

The three patients showed interesting differences in emotional states. The first was extremely anxious, emotional and worried about her condition. The second was optimistic and wanted to get well. The boy was apathetic, had no interest in his condition, and in general showed the mental state usually found in such cases, which sometimes causes the patients to be classed as hysterias.

Dr. Smith Ely Jelliffe thought it was unique to have three members of a family showing the beginning, middle and developed phases as had been demonstrated. Dr. Abrahamson had mentioned Ziehen's familial group, which Dr. Jelliffe had had the good fortune to study in Berlin. There were three in that group also, and Schwalbe's monograph had presented the features, but in Ziehen's group the disorder had progressed to a more or less uniform pattern and no developmental study was possible such as the present presentation offered.

Dr. Jelliffe was disposed to emphasize the varying clinical trends of a large group of striatum syndromes of which these cases were but one of the striking types. It had become increasingly evident that dystonia musculorum, Vogt's double athetosis, Westphal's pseudosclerosis, Wilson's lenticular degeneration, Huntington's chorea, tuberous sclerotic idiocy, and even paralysis agitans were to be regarded as but variants in this larger picture of striatum syndromes.

It was recalled that one of Ziehen's patients had come to necropsy and negative findings had been reported, but more recently one of Flatau and Sterling's cases had been examined postmortem, and Thomalla,[1] Schneider[2] and von Economo[3] had respectively studied the striatum pathology and the liver, for the case of Flatau and Sterling had afforded a combination of the Ziehen-Oppenheim group of dystonia musculorum types and Wilson's lenticular degeneration types, since the clinical picture was characteristic of the former trend, while the hypertrophic sclerosis of the liver was of the Wilson lenticular degeneration type. The pathologic picture of the striatum was one of abiotrophic atrophy of the cells of the putamen.

Vogt[4] and Bielschowsky[5] quite recently attempted a synthesis of the various striatum syndromes outlining the pathologic trends and enlarging the neurologic pictures to be comprised in the group. Thus the Vogts distinguish (1) the *état marbé,* which they originally described as double athetosis and in which group many instances of Little's disease are to be included; (2) the *état fibreux,* which progresses often as a bilateral progressive chorea without psychic symptoms, also shown in Huntington's chorea; (3) total necrosis of the striatum, chiefly confined to the putamen, and at times reaching to other parts of the globus pallidus. To this group the dystonia case of Thomalla belongs and Wilson's lenticular degeneration. (4) Acute infectious striatum syndromes; many of these have been observed in our recent lethargic encephalitis epidemic as paralysis-agitans-like cases, athetotic cases, pseudobulbar palsies, involuntary laughing and crying cases, etc. It would lead too far to take up Bielschowsky's grouping of the various syndromes, and Dr. Jelliffe referred to these recent studies.

One point of speculative interest Dr. Jelliffe said he would like to raise, namely, the opportunity here offered to study the syndromes now under discussion and their relationships and that of the striatum to the vegetative control of muscle metabolism through the liver. He spoke of correspondence with Wilson relative to the evidence afforded by lenticular degeneration and liver metabolism. Whereas Wilson had argued for a toxin from perverted liver function as causing the striatum changes, Jelliffe would emphasize the neural integration disturbance of striatum interruption of vegetative liver pathways. The case of Thomalla, in which the liver was carefully studied by Schneider, offered several interesting points bearing on the possibilities of an analysis of the vegetative integration mechanism which was essential for the liver function, interference with which caused the vagotonic gliosis, and this put the entire muscular metabolic functions to disadvantage, in that the integration of the involuntary part of the muscle structure, the sarcoplastic substance and the

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1. Thomalla: Ztschr. f. d. ges. Neurol. u. Psychiat. **41**: 1918.
2. Schneider: Ztschr. f. d. ges. Neurol. u. Psychiat.
3. Von Economo: Ztschr. f. d. ges. Neurol. u. Psychiat. **43**:173, 1918.
4. Vogt: Jour. f. Psychol. u. Neurol. **24**:1, 1918.
5. Bielschowsky: Idem. **24**:20 and 28, 1918.

sensori motor anisotropic disk parts were seriously impaired. Here was an important field in which future studies could be prosecuted and the vegetative arc components, their spinal pathways and higher integrative synaptic connections laid bare.

Dr. Tom A. Willaims of Washington (by invitation) spoke of the physical components of muscle and the mechanism of muscular tonus. The chemistry of muscle reaction and phases of metabolism have been dealt with by Bjerron.

These cases, Dr. Williams felt, did not show true propulsion, since they were able to arrest themselves and turn without difficulty. The attitude assumed by the second patient in walking caused a sort of propulsion. The boy, however, could go upstairs and walk and run. The peculiarity of gait is a hypotonus rather than a true propulsion. Wherever there was voluntary increased tonus the propulsive phenomena would seem to disappear. In other words, the phenomena observed might be termed pseudopropulsion due to dystonia, overcome by volition.

Dr. Abrahamson, in closing, explained that clinical propulsion, such as was seen in Parkinson's diseases and in certain cerebellar affections, was dependent on the abnormal attitude of the patients or to a faulty gait.

True propulsion or forced movements resulting in propulsion were to be found in lesions affecting the pathways subserving static equilibrium, i. e., the semicircular canals — vestibular nerves, Deiter's, Bechterew nuclei, postlongitudinal bundles or interstitial nucleus of the thalamus. Experimental work on this pathway, in particular the work of Muskens on the posterior longitudinal bundle, proved this. In the cases of dystonia shown the patients exhibited the clinical type of propulsion and not the forced movement type.

AN ANALYSIS OF THE CASES ADMITTED TO THE NEUROPSYCHIATRIC SERVICES OF THE U. S. ARMY GENERAL HOSPITAL NO. 1 (COLUMBIA WAR HOSPITAL, NEW YORK CITY).

Dr. Sylvester R. Leahy.

This article will be published in full in the Archives.

ACUTE DESCENDING RADICULITIS — A SPINAL TYPE OF EPIDEMIC ENCEPHALITIS. Dr. Irving H. Pardee.

This article is published in full in this issue.

A STUDY OF PUBERTAS PRAECOX AND THE MENTALITY OF THE PATIENTS. Dr. J. H. Leiner.

Dr. Leiner reviewed the historical references to pubertas praecox. In this syndrome it would seem that the child passed through several stages of life in utero. The endocrines seem to be a primary factor in the causation of the condition. Secondary factors are climate, race and heredity. As is well known, menstruation appears normally at a somewhat earlier age among women in southern countries, while the inhabitants of the north normally do not begin to menstruate until after the age of 14 or 16. Marriage in Oriental races takes place early, and the precocity of the southern races may be due to inbreeding.

Dr. Leiner described two cases in which there had apparently been direct hereditary transmission. One patient, a girl, at birth gave the impression of a 12-year-old child; menstruation began at 6 weeks and was regular thereafter. A second patient, a boy, at 4 years of age looked as though he were at least 10, and had the physical development of a young man of 21. The parents of both of these patients reported marked virility, or actual instances of pubertas praecox in one parent.

Precocious puberty is caused by a hypersecretion of either the gonads, pineal or cortico-suprarenal glands. A secondary involvement of the pituitary and thyroid is also unquestionable. Clinicopathologic evidence shows that the first three glands are involved in this syndrome, either in the form of hyperplasia affecting them or neoplasms.

Rogers collected 101 cases of pubertas praecox, eighty-one cases in the female, and twenty in the male; of the eighty-one cases, seventy-three point to the hyperovarian type. A number of other writers have recorded cases of this type, among them Lenz, who described the case of a girl in whom menstruation began at 16 weeks. The secondary sex characteristics were those of a mature woman. As she grew up she became a good scholar, but preferred the society of children of her own age. At 12 she was shy and childish in behavior.

Lucas reports neoplasm of the ovary as a cause of pubertas praecox. At 7 his patient showed all the signs of genitosomatic maturity, with early menses. After removal of a tumor of the ovary, all signs of adolescence and menstruation disappeared. Eleven cases of sexual precocity associated with ovarian neoplasms were collected by Roger Williams. This does not necessarily indicate that tumors of the ovary lead to sexual precocity, since other factors enter into the causation. The mentality in the ovarian cases never seems to be great, in fact, the patients speak and act their true age. Early menstruation in hyperovarianism produces excess calcium elimination. This results in short stature.

In the cortico-suprarenal type of cases the clinical picture differs according to whether the involvement is in the male or the female. Hyperplasia of the suprarenals in the male tends to accentuate male precocity; in the female the tendency is to change the female into the male type with all the secondary sexual characteristics of the male. About fourteen cases of this hypertrichosis of the male type manifested a large clitoris, complete absence of menstruation, and frequently hair on the face. The mentality in these cases is low. A certain degree of aggressiveness is reported.

In cases of hypergonadal condition in the male, mentality is usually retarded. In one case of precocious sexuality the removal of a malignant tumor of the testicle caused the disappearance of the adult characteristics.

Tumors of the pineal and their effect on sexual precocity have been extensively studied. As yet no direct connection seems to have been demonstrated, aside from the statistical fact that pineal tumors occur predominantly in the male, while those of the suprarenals are most frequent in the female.

There is little or no real mental precocity in all these types; the patients are usually shy and reserved on account of their appreciation of their differences from the normal type. Early diagnosis in the hyperplastic types may result in improvement by proper endocrine therapy.

Dr. Walter Timme added that two cases, as yet not published, had come to his attention. They were both in the same family. One of the two girls was then 16, had begun menstruating at 5 and had developed a psychosis of the dementia praecox type. The younger sister was 3 years old, menstruation had begun before 2, the child was of the incorrigible type and was apparently advanced in intelligence. Both showed pineal shadows, which are to be observed with extreme rarity at these ages. The locus of the disturbance was probably in the pineal.

With reference to Dr. Leiner's comment on the relation between skeletal growth and menstruation, Dr. Timme knew of cases that would controvert the opinion that late menstruation was accompanied by increased height. In a case that he knew, although menstruation began after 17, the patient remained small, and the epiphyses were ununited. The patient was treated on the basis of retarded growth; endocrine therapy produced rapid growth.

AN UNUSUAL CASE OF EPIDEMIC ENCEPHALOMYELITIS. Presented by Dr. Walter M. Kraus.

J. B., a man, aged 32, a riveter, was admitted to Bellevue Hospital on Feb. 23, 1920, complaining of pains and weakness in the shoulders and arms. For three or four weeks prior to admission he had had pains in the shoulders and arms. This was increased by movements. Soon after the onset, weakness of the upper extremities became noticeable and finally compelled the patient to stop work on February 10. He noticed diplopia one week before admission to the hospital.

On admission there was weakness and tenderness of the muscles of both arms from the deltoids down. There were fibrillary twitchings in the right deltoid. There was marked winging of the right scapula (paralysis of the long respiratory nerve of Bell to the serratus magnus). Some winging of the left scapula was also present. The weakness was generally greater on the right than on the left. This may have been due to the fact that for twenty years the patient had been accustomed to carry heavy pieces of iron on the right shoulder. The pectoral muscles were strong. There was a slight weakness of the muscles supplied by the left seventh cranial nerve, and a mask-like expression. Nystagmoid movements of both eyes to right and left were observed. Tremor of the eyelids, tongue and hands was present. Reflexes: The triceps reflexes were absent; biceps ++.R$<$L. Supinator reflexes were present, right and left, and the other reflexes were normal. There were no sensory changes beyond the pain noted and no incoordination.

Progress: April 26: Diplopia still present. March 1: Sleepiness was marked and hard to control. March 4: Atrophy and tenderness of both intraspinati noted; white blood cells, 10,400; polymorphonuclears, 60 per cent. March 13: The tenderness in the shoulders had gradually disappeared and there was now tenderness in the hands. March 17: Pill-rolling type of tremor noticed in both hands. March 18: The gait was shuffling. Conjugate movement of both eyes downward was poor.

Laboratory Findings: Spinal fluid on admission: 40 cells; globulin +; colloidal gold curve 0000121000. The Wassermann reaction was negative.

Present Status: Slight left facial weakness; complete paralysis of the right serratus magnus and partial paralysis of the left serratus magnus (winging). Electrical Reaction: Complete reaction of degeneration in the right serratus magnus. All the other muscles of both arms, forearms and hands showed a partial reaction of degeneration. There were fibrillary tremors, atrophy and weakness of all the muscles of both upper and lower extremities. In brief: A case of acute epidemic encephalomyelitis showing among other signs the results of involvement of the anterior horn cells ofthe lower cervical (5, 6, 7, 8) and first thoracic spinal segments.

Book Review

APHASIA AND ASSOCIATED SPEECH DEFECTS. By MICHAEL OSNATO, M.D., Associate in Neurology, Columbia University. Paul B. Hoeber, New York, 1920. 183 pages.

The author of this little book is not satisfied with the doctrine of the localization of speech as that doctrine has been understood for a generation or so, and sets out to demolish it. His argument rests on two legs. First, the postmortem findings in some cases of aphasia have not supported the claims of the localizationists. Thus, as is well known, some cases of motor aphasia have been reported in which no lesion in Broca's area had been observed. This negative proof seems to make a strong appeal to Dr. Osnato, in spite of the fact that negative proof is always open to suspicion. He makes little attempt to meet the mass of positive proof which supports the main doctrine of motor and sensory aphasia, except by reference to a few statistical papers by other workers. His own work is entirely lacking in anatomic studies. But statistical studies are usually made by the aid of the midnight oil; they are not original studies when based on papers written by others, and therefore are not of first hand value. They admit of all sorts of personal interpretation and prejudice. Anything can be proved by statistics.

The gist of the book is, of course, not in its negative or destructive criticism, but in the thesis which Dr. Osnato himself advances for the physiology of speech. He frankly returns to the antique doctrine that in the functioning of speech the brain acts as a whole. It goes off somewhat like a Leyden jar. This, we believe, or something like it, used to be the doctrine of Flourens and his school in years long gone. But Dr. Osnato marshals his pathologic data, and these he has borrowed largely from such affections as dementia praecox and other organic dementias. Using Southard's work in dementia praecox, he points out that although these patients presented speech defects, including aphasia and its associated apraxia, nevertheless the lesions in the cortex were more or less diffused and not localized in the so-called speech centers. Ergo, there is nothing in cerebral localization of speech. This argument may appeal to some emancipated critics, but it will not appeal to everybody. The simple and obvious objection to it is, of course, that a widely diffused lesion is incapable of proving anything, either pro or con, on such a subject as cerebral localization. The only proof that can stand is proof based on localized lesions —and such lesions in sufficient numbers are now on record to support the claims of the localizationists.

Dr. Osnato, summing up his opinions on page 135, repeats that "the function of speech and voluntary movement is an integral part of the development of the functions of the entire brain, and is incapable of being anatomically, physiologically or psychologically separated into centers." This is the whole book in a nutshell. But the writer of this review must enter a mild protest against this summary dismissal of all that we thought we had learned about cerebral localization. Let others judge for themselves of the value of Dr. Osnato's arguments by reading his book. It will entertain, even if it does not convince them.

The American Medical Association will pay 50c each for the April and May, 1919, issues of the Archives of Neurology and Psychiatry. Address to American Medical Association, 535 North Dearborn St., Chicago, Ill.

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SECTION OF THE ANTEROLATERAL COLUMNS OF THE SPINAL CORD FOR THE RELIEF OF PAIN

A REPORT OF SIX CASES *

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It is the surgeon's duty at times to perform operations conceded to be only palliative in effect, such as gastro-enterostomy for incurable gastric carcinoma, tracheotomy for a malignant lesion of the larynx, subtemporal decompression for inoperable tumors of the brain. When pain is a conspicuous feature, justification for operation is undeniable. I have, on more than one occasion, removed the gasserian ganglion for an inoperable tumor of the middle fossa. The suffering, attending lesions of the spine or spinal cord involving the posterior roots, is often beyond relief from opium or its derivatives and under such circumstances surgical measures may with propriety be considered. In 1912 Spiller proposed section of the anterolateral columns of the cord, when for one cause or another the patient is suffering from intractable pain. Since that time I have carried out this operation on six occasions; in four there was complete, and in two partial, relief.

Based on his studies of the location of pain fibers within the spinal column, Spiller[1] was led to the conclusion that fibers for the conduction of pain were located in the anterolateral columns of the cord, and it was on this observation that he proposed the operation under discussion. Grouped with the fibers that convey pain are those for temperature sense, so that it must be taken for granted that section of the anterolateral columns implies not only loss of pain, but also of thermal sense. The loss of perception of heat and cold in one or both hands might be regarded as serious and might be advanced as an argument against the operation in question. As the section of the column would not, under any circumstances, be made high enough to include the brachial plexus, this objection is not to the point. Loss of thermal perception in the trunk and lower extremity is relatively unimportant and would gladly be exchanged for pain by the patient.

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* Read at the Forty-Sixth Annual Meeting of the American Neurological Association, June, 1920.

[1] Spiller: Univ. Penn. M. Bull., July and August, 1905.

The anterolateral column cannot be sectioned without including Gower's tract, and one might question whether objective disturbance of sensation would follow the operation, either as a temporary or persistent sequel. The character of the cases on which we operated, the presence, in some before operation, of disturbances of sensation, prevented enlightening observations on this point, save in one case, and in this tactile sensation was preserved throughout where pain and thermal sensation were absent.

In this connection I recall the experiments of Cadwalader and Sweet,[2] who, after section of the anterolateral columns in dogs, noted in addition to the loss of cutaneous sensations for pain and heat, a pronounced ataxia. This ataxia was attributed to injury to Gower's tract, and Cadwalader looked on this as a pseudoparalytic rather than a true paralytic ataxia, as part of profound disarrangement of the pseudomuscular apparatus. The sensory impression that should pass through Gower's tract had been cut off and, as the compensatory function of the remaining sensory tracts was being established, the pseudoparalysis gradually disappeared. Very briefly, these are what might be called the theoretical considerations. Let us now review their practical application.

My experience with this operation includes a series of six cases, in three of which the source of pain was a malignant lesion, and in three gunshot wounds of the spinal cord or lumbosacral plexus.

REPORT OF CASES

CASE 1.—*Summary.*—The patient had an inoperable sarcoma of the spine at the level of the second and third lumbar vertebrae. Suffering was intense and uncontrolled by morphin. Chordotomy was employed with section of the anterolateral columns, right and left, at the level of the first lumbar segment. This resulted in total loss of thermal and pain sensation.

History.—M. B., aged 31 (File No. 33336), was admitted to the University Hospital Aug. 25, 1916. Seven months before the patient began to complain of pain in the left knee and three weeks later of pain in the right knee. Later he experienced pain across the lower end of the sacrum and severe burning sensations from the tuberosity of the ischium to the knee. When admitted to the hospital he had edema of both feet, and there was almost complete paralysis of the left and partial paralysis of the right lower extremity. The tendon reflexes of both extremities were abolished. There was no disturbance of tactile, pain or temperature sense. The patient complained bitterly of burning pain, starting at the hips and radiating to the knees and of cramp-like pain in the calves.

Exploratory Laminectomy, Oct. 7, 1916.—The spinous processes of the first, second and third lumbar vertebrae were removed. At the level of the third lumbar lamina a large cavity was discovered, extending up and down the left side of the vertebral canal, containing a mass of tumor tissue. It was evident from its nature and extent that the tumor was inoperable.

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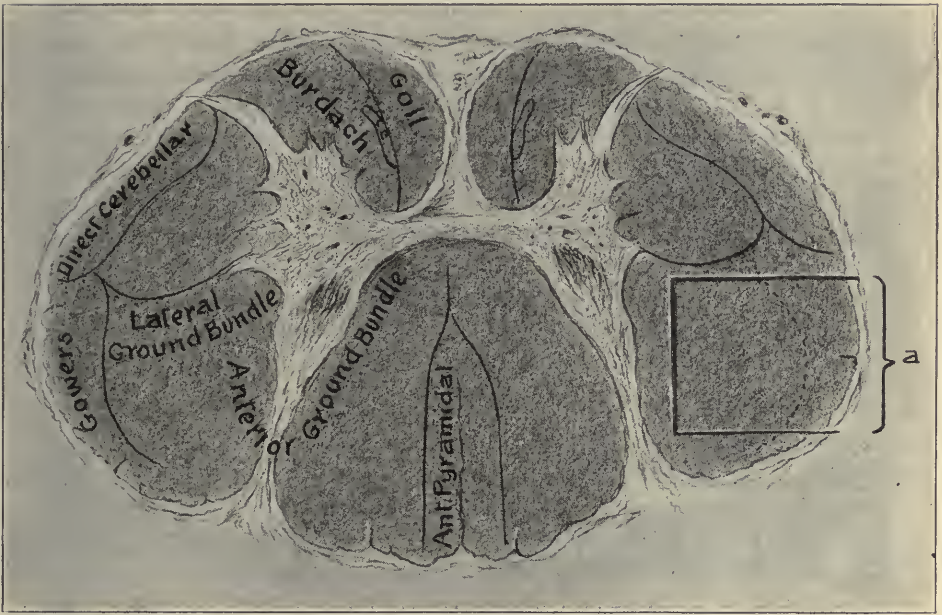
[2] Cadwalader, W. B., and Sweet, J. E.: Experimental Work on the Function of the Anterolateral Column of the Spinal Cord, J. A. M. A. **58**:1490 (May 18) 1912.

Pathologic Report.—A small round cell sarcoma was found.

The insufferable pain continued, it was uncontrolled by morphin, and for its relief it was decided to do a chordotomy. Before the operation an examination was made of the disturbance of sensation in the lower extremities.

Chordotomy, Nov. 11, 1916.—Ether anesthesia was used. The spinous processes and laminae of the ninth and tenth thoracic vertebrae were removed. A pledget of cotton, saturated with 0.4 per cent. stovain, was applied to the surface of the cord and roots above the level of the section (stovain block). The cord was rotated by traction on the denticulate ligament. The anterolateral column was sectioned 3 mm. by 2.5 mm., first on the left then on the right side. The wound was closed with tier sutures.



The spinal cord at the level of the sixth thoracic segment. Cutting the anterolateral columns of the cord, as described, implies section of that portion of the cord included in the area marked *a.*

Nov. 16, 1916: Five days after the operation, an examination of sensory disturbance revealed practically complete loss of pain and thermal sensation in the area below that supplied by the first lumbar segment.

Eleven days after the operation the patient's condition had much improved, he was able to sit in a wheel chair, whereas before the operation he was bedridden and his condition pitiable.

Six weeks after the operation the patient was discharged from the hospital. He had continued free from pain.

Commentary.—I know of no other conditions in which the pain is more intense than in malignant tumors of the vertebral column, whether in sarcomas or carcinomas. The invasion of the posterior roots by the tumor, as the source of pain, suggests the possibility of relief by a rhizotomy or division of the posterior roots. As a matter of fact, on several occasions I have resorted to this method. But section of the anterolateral columns is, I think, to be preferred to rhizotomy. As a theoretical objection to rhizotomy in inoperable tumors of the vertebrae one has but to consider the likelihood of return of pain, as the tumor increases in size and invades the roots above or below those divided at the operation. There is another reason, I think, for preference being given to chordotomy — chordotomy is easier of execution than rhizotomy. No matter what the lesion is or where it is located, the operation implies the exposure of but one spinal segment. This is not the case with rhizotomy, in which a series of roots may have to be divided and a corresponding number of segments exposed. And then no matter where the lesion is located the operator may select for his own convenience the segment at which the cord section is to be made. Latterly, I have chosen the sixth cervical segment as being in a region of the vertebral column at the apex of its normal convexity and where it is the more readily accessible. It is a matter of no little consequence that in an operation de complaisance the operation should be stripped of all technical difficulties and thus freed from risks. There have been no fatalities in our series.

Case 2.—*Summary.*—The patient had a gunshot wound of the spine with intense pain in the left lower extremity. Four grains of morphin a day were given with only partial relief. Unilateral chordotomy was performed and resulted in complete cessation of pain. There had been no recurrence at the last observation twelve months later.

History.—W. P., a young man, aged 21, received a gunshot wound in the lower dorsal vertebrae. Several months had elapsed from the time of the accident until he came under the writer's observation. At that time he had a complete paraplegia and was suffering day and night from pain which radiated down the left thigh. To control the pain, hypodermics of morphin, as circumstances might require, were prescribed. As 4 grains in the twenty-four hours gave only partial relief, operation seemed justifiable.

Dec. 12, 1918: Chordotomy was performed; also laminectomy of the fifth and sixth thoracic vertebrae, and section of the anterolateral columns at the level of the sixth cervical segment. The wound was closed with tier sutures.

Postoperative Course.—The convalescence was uneventful. The results were brilliant. The patient was given one hypodermic the night of the operation and after that none. The pain disappeared, and when last seen, twelve months later, there had been no recurrence.

Commentary.—The indications for some radical measure to relieve pain were in this case commanding. The patient was in a state of demoralization and exhaustion from suffering day and night. He was confined to bed because body movement excited pain. Although given morphin freely and in increasing doses he was afforded only a measure of relief. The immediate withdrawal of morphin after operation from a patient addicted to large doses, was a striking and assuring demonstration of freedom from pain. The patient gained in weight, he was soon able to go about in a wheel chair, and he was transferred from a person in abject misery to a reasonably cheerful creature, despite his other disabilities.

Case 3.—*Summary.*—The patient had carcinoma of the rectum, with pain in the rectum, buttock and thighs. Colostomy and section of the anterolateral column, left and right, were performed. Complete loss of thermal and pain sense on one side only resulted. Radium therapy was employed for the carcinoma. Four years later the patient reported himself as free from discomfort; presumptive recovery.

History.—L. W. G., a man, aged 44 (File No. 32063), was admitted to the University Hospital May 24, 1916. His case was diagnosed as carcinoma of the rectum. A large mass, just above the anal canal, filled the lumen of the bowel. The condition was inoperable.

Treatment and Course.—May 25, 1916: Colostomy through the left rectus muscle, under local anesthesia, was performed.

June 1 and June 21: Ninety mg. of radium were applied for twenty-four hours. On June 23 he was discharged from the hospital. His general condition had improved. The colostomy was functionating well. He continued to complain of pain in the rectum, perineum and legs.

Sept. 22 and Nov. 23, 1916: The patient was readmitted to the hospital for radium therapy. His condition was one of demoralization. He was complaining bitterly of pain in the rectum, perineum and thighs. Large doses of morphin gave only partial relief.

Jan. 18, 1917: Chordotomy was performed under nitrous oxid anesthesia. The spinous processes of the ninth and tenth thoracic vertebrae were removed. Section was made of the anterolateral column on the left side. The cataract knife entered the cord 2.5 mm. posterior to the point at which the anterior root leaves the cord, and section of the cord was made from this point forward 2.5 mm. and 2.5 mm. deep. The anesthetic was discontinued and an attempt was made to test sensation, but the patient was too disturbed and restless. The anesthesia was resumed and preparations made to section the anterolateral column on the right side. Section of the anterolateral column on the left side seemed to be in accordance with the prescribed measurements, but on the right side it was the opinion of the operator that the section was not liberal enough.

Jan. 25, 1917: One week after operation there was a flaccid paralysis of the left lower extremity, and both the patella tendon and Achilles tendon reflexes were diminished. There was no motor disturbance of the right lower extremity and the reflexes were normal. The patient complained of bearing down pain in the bladder.

Feb. 2, 1917: A physical examination was made which revealed: Right lower extremity: There was preservation of tactile sensation throughout and loss of thermal and pain sensation over the buttock, perineum, posterior aspect of the thigh and below the level of the knee. There was no disturbance of sense of position. Movements of the limb were unimpaired. The patella and Achilles' tendon and cremasteric reflexes were normal. There were abortive ankle clonus and dorsiflexion of the great toe.

Left Lower Extremity: The patient could distinguish between hot and cold and the prick of a pin throughout the extremity. The sense of touch was preserved, although the patient stated that sensation was not so acute in the anterior and lateral surfaces of the thigh. There was no disturbance in the sense of position. All movements of the left lower extremity, temporarily arrested after the operation, have been restored. Achilles and patella tendon reflexes were more active than at the last examination, January 25. Ankle clonus and the Babinski reflexes were present. The patient continued to have attacks of vesical tenesmus, but they were not so frequent or so painful as formerly.

April 21, 1917: The patient was discharged. There had been no change in the sensory disturbances as noted Feb. 2, 1917. Several attempts were made to have the patient return for continued radium treatments, but without success. During the war he was lost sight of.

Sept. 10, 1919: The patient writes "I do not feel any soreness in the perineum or rectum. I ride a bicycle a great deal without discomfort. My appetite and digestion are good, and my limbs are strong."

May 23, 1920: Four years after the patient came under observation his condition was reported through a verbal communication as satisfactory.

Commentary.—This is an extremely interesting case from many points of view. Too often we relegate to the scrap heap patients with apparently incurable lesions. Nothing could have appeared so utterly hopeless as this huge rectal carcinoma. Four years have elapsed since treatment was instituted and the patient once bedridden, is up and at work. The peculiar susceptibility of rectal carcinoma to radium therapy is, of course, well known, but the symptoms, when the patient was first seen, were at least suggestive of extension of the tumor beyond its original site. I am reminded, in speaking of seemingly hopeless cases, of a case of inoperable sarcoma of the spine. To relieve pain I divided a series of posterior roots and instituted roentgen-ray treatment and injections of Coley's fluid. Three years and three months have elapsed since the operation, and when the last roentgen-ray treatment was made the tumor shadow had entirely disappeared. Returning to the case of rectal carcinoma, it was evident that one side of the anterolateral column was not entirely divided, at least not sufficiently, since perception of pin prick and heat and cold in the left lower extremity were not abolished altogether, as they were in the right lower extremity. As commented on elsewhere, there is, of course, a possibility of error on the part of the operator in including the prescribed amount of tissue in this section.

Case 4.—*Summary.*—The patient had an inoperable sarcoma of the thigh with pelvic metastasis and insufferable pain in the gluteal region. The anterolateral columns of the cord were sectioned with relief from pain from the time of the operation until the patient's death, months later.

History.—T. C., a man, aged 39 (File No. 38089), was admitted to the University Hospital Dec. 22, 1918. In August, 1916, he noticed a sharp pain in the thigh. The latter increased in size and a diagnosis of sarcoma was made. Two operations were performed for the removal of the growth by Dr. J. H. Page, Austin, Pa., Aug. 28 and Oct. 3, 1917. One month later there was evidence of recurrence.

Examination.—Examination on admission revealed a tremendous infiltrating diffuse tumor of the right thigh, edema of the leg, dilatation of the superficial veins and enlargement of the inguinal lymph nodes.

Pathologic Report.—The pathologic report of tissue removed for examination stated that perithelial small round cell sarcoma had been found.

Treatment and Course.—Treatment with Coley's fluid and radium was instituted. The patient complained bitterly of pain referred chiefly to the gluteal region, and while the lesion was inoperable, the distress of the patient justified some attempt to relieve the pain.

Jan. 30, 1918: Chordotomy was performed and spinous processes and laminae of the seventh and eighth thoracic vertebrae were removed. The denticulate ligament was grasped with mosquito forceps, the cord rotated and, with a sharp curved needle, armed with fine silk, the anterolateral column was encircled, and with the cord thus fixed the column was divided with a cataract knife.

Postoperative Course.—The patient made an uneventful recovery. He was entirely relieved of pain and continued free from pain until his death several months later.

Commentary.—The condition of the patient, in the judgment of the operator, justified some radical means of relief. The propriety of any operation under conditions in which the expectation of life is brief, might be questioned, but I should have no hesitancy under similar circumstances in recommending a chordotomy, especially as no risk is entailed and it assures the patient an extraordinary measure of relief from pain for the months that remain to him.

Case 5.—*Summary.*—The patient had received a gunshot wound of the spine, and suffered pain in both lower extremities. Chordotomy was performed with loss of pain and temperature sense.

History.—H. G., a man, was operated on at a U. S. Army General Hospital, March 3, 1919. The patient was injured overseas, and complained of shooting pains in both lower extremities. These were of such severity as to justify an attempt at relief by operation.

March 3, 1919, a chordotomy was performed. The sixth thoracic segment was uncovered by removing the laminae of the fifth and sixth thoracic vertebrae. Immediately after section of both anterolateral columns sensation was tested by Dr. Cobb and there was complete loss of pain and temperature sense up to the level of the umbilicus on both sides. The patient survived the operation. (The clinical record of the subsequent course is not available.)

Case 6.—*Summary.*—The patient had received a shell wound of the pelvis with injury to the sciatic nerve. He had had intense unbearable pain in the thigh and foot for two and a half years, unrelieved and unabated. The anterolateral columns were sectioned with absolute relief. There was recurrence of pain, much more moderate, several months later.

History.—H. E. H. D., a man, aged 50 (File No. 58567), was admitted to the University Hospital May 12, 1919, under the care of Dr. W. G. Spiller. He had been wounded in France, Oct. 21, 1916, by a high explosive shell which penetrated the pelvis on the right side below the crest of the ilium. Five weeks after the injury he began to complain of pain in the right leg, radiating down the course of the sciatic nerve to the foot. The pain in the right foot never ceased; it was worse at night, and was shooting and burning in character. The pain in the thigh was of like character, and began about 12 inches above the knee and extended 3 inches above the midpopliteal space.

The patient in walking was unable to put the right foot to the ground and always used crutches.

Examination.—May 14, 1919: Touch, pain and thermal sensations were lost in the right sciatic distribution. In the popliteal space on the inner side of the leg and on the inner side of the sole of the foot there appeared to be some sensation, probably from the overlapping of adjacent nerve territories. The right calf and right thigh were much wasted. He could flex the right leg at the knee to a right angle with fair amount of power, but resistance to passive movement showed great weakness in the flexors of the leg on the thigh. He had a little dorsal and plantar flexion of the right foot. The sole and heel showed evidences of trophic disturbance in the thickening and desquamation of the skin. The patellar reflex was about normal on the left side, and was present but diminished on the right. The Achilles tendon reflex was normal on the left, but absent on the right side. Frequent involuntary jerking of the right lower limb followed twinges of pain (Spiller).

Roentgen-Ray Examination.—The shell evidently injured the upper portion of the ilium and the transverse process of the fifth lumbar vertebra. There was evidence of a preexisting osteomyelitis in the projections of bone which indicated calcification along healed sinuses. The lesion was evidently outside the vertebral column.

Operation, Treatment and Course.—A chordotomy operation was performed by Dr. C. H. Frazier, May 24, 1919. The spinous processes and the laminae of the sixth and seventh thoracic vertebrae were removed. Rotation of cord was accomplished by traction on denticulate ligament. One anterolateral column of the cord was sectioned. Closure of the dural incision was made with continuous silk suture. The wound was closed with tier sutures.

Postoperative Examination, June 10, 1919: Sixteen days after operation the following notes were recorded by Dr. Spiller:

"Tactile, heat and cold and pain sensations are greatly impaired down the back of the thigh, the outer side of the leg beginning about the union of the upper with the middle third, in the dorsum and sole of the right foot except at the inner margin. Cold sensation is sometimes called hot and is always recognized in an uncertain manner on the inner side of the right leg, front and sides of the right thigh and right side of the abdomen, about one or two inches above the umbilicus. Here the impairment of temperature sensation shades off gradually into the normal area. This impairment of temperature sensation is pronounced in the right lower limb but never amounts to a complete loss. Pain sensation is diminished in the right lower limb and right lower abdomen, as is temperature reaction. A moderate prick of the pin which would be recognized promptly in the left side is often called dull. There is therefore a positive diminution in pain sensation in the right lower limb, which never amounts to a complete loss.

"Localization of touch is as good or nearly as good in the right lower limb as in the left. The toes of the right foot are so rigid from the result of an old injury to the foot that sense of position cannot be properly tested.

"He still has paresthesias which he describes as if the part were asleep. This is confined entirely to the sole of the right foot. It is probable that this paresthesia is the result of the old injury to the nerves of the foot and is not the result of the recent operation. The pain of the right foot was so intense before the recent operation that it probably obscured any paresthesia that might have been present."

From the time of the operation to the patient's discharge from the hospital, June 19, 1919, the patient was practically free from pain. This was the first time he had enjoyed any relief since the onset of pain two and a half years ago. July 4, 1919, after a trip cross the continent, the patient wrote: "I stood the trip very well and feel that I have taken a new lease of life. Since the operation or a day or so after, I have had no pain at all, although I have a peculiar sensation in my foot most of the time. This I described to Dr. Spiller, and is perhaps more noticeable now probably because *I am using my foot a good deal. However, this is not a pain and really bothers me very little.*"

Jan. 8, 1920, word was received that during the past three months there had been a return of pain, but it was reported to be not nearly as severe as before the operation.

Commentary.—This was unquestionably a case in which a chordotomy was indicated. The pain was unbearable and the patient was in great distress for two and a half years. The nature of the lesion was conjectural, possibly a nerve compression or neuroma, but the location of the lesion within the pelvis was such as to make it difficult of approach and it was doubtful whether a nerve liberation would have given relief. At all events, after explaining to the patient the difficulty attending an operation, designed to explore the lesion, and presenting the alternative proposal, a chordotomy, he chose the latter.

The immediate and complete relief which followed the operation was a practical demonstration of what may be expected when the anterolateral column is divided. That there should have been recurrence of pain, though not in its original severity, illustrates the only difficult problem in the performance of the operation. It is, of course, difficult to make the section of the cord according to prescribed measurements. The anterolateral column is estimated as 2.5 mm. in width and depth. To make the section with mathematical precision and without injury to adjacent structures is not an easy matter, but I believe with the introduction of instruments particularly designed for this purpose, fewer errors will be made, and the percentage of complete and permanent recoveries will be higher. The operation is founded on sound physiologic principles, and it remains only to overcome this technical difficulty.

CONSIDERATIONS OF TECHNIC

The success or failure of this operation is wholly dependent on the accuracy with which the operator follows his instructions. There are two points to be considered: 1. At what level shall the section of the anterolateral column be made? 2. How shall the section be made?

By way of preface it may be said that the cord should be exposed by the removal of the spinous processes and the laminae of two vertebrae. At least two spinous processes should be removed, and I think it is better to remove two rather than one lamina. One needs amplitude of exposure in all intraspinal procedures, and for this reason I disapprove of unilateral laminectomy. The removal of but half the arch does not give sufficient exposure and has no merits whatsoever.

1. The question of segmental selection is a matter of some consideration. We assume that the lesion does not involve the upper extremity. Manifestly, the section could not be made sufficiently high, that is, several segments above the origin of the brachial plexus, with any degree of safety. We assume that the lesion is in the pelvis or involves the spinal column not above the upper thoracic vertebrae. With this understanding, a general rule might be laid down that the section should be made at least four segments above the lesion so as to ensure the crossing of all afferent tracts and to allow for the extension of the growth upward — it would be well to add two or three segments. So much for the physiologic or anatomic grounds. From the practical or technical point of view, I have selected the sixth thoracic segment as the most convenient for all lesions not higher than the ninth or tenth segments. (As a matter of fact, in all my cases, the lesion has been below this level.) The fifth and sixth thoracic vertebrae or their spinous processes are near the apex of the normal convexity of the spinal column and the spinal cord at this level is nearer the body surface and therefore more accessible. For this reason let us consider that the sixth thoracic segment is the site of election.

2. To answer the question—How shall the section be made?—we must recall the topographical relationship of the columns in the lateral aspect of the cord between the points at which the anterior and posterior roots are given off. If we should divide the distance between the posterior and anterior roots in two, that area of the cord dorsal to the dividing line contains the direct cerebellar and lateral pyramidal tracts; that ventral to this line contains the lateral and anterolateral tracts and Gower's tract. It is this area of the cord with which we are chiefly concerned.

To include the anterolateral tract in the section, it is necessary of course to sacrifice Gower's tract, but from such observations as we have been able to make, this does not lead to impairment of tactile sensation. The crux of the situation is this: How wide and how deep must the section be made? As to width, the posterior boundary would be represented by a point midway between the anterior and posterior roots. In the past we have divided the tissue between this point and the anterior root, but Spiller is disposed to believe that one could include with propriety the anterior root in order to assure division of the entire tract. This, I think, is an excellent suggestion, since one anterior root at this level might be sacrificed with impunity. Of greater difficulty is the determination as to depth. The estimated distance from the surface of the cord to, and including, the anterolateral column is 2.5 mm., but in some of our cases there has not been total loss of pain and thermal sense. Whether this was due to inaccurate execution at the operation, or to underestimation of the dimensions of the column is a matter for discussion. I am inclined to attribute our failures to the former rather than to the latter, since in some instances there has been total loss of thermal and pain sense when the knife penetrated the cord the prescribed distance, i. e., 2.5 mm.

A word might be said in conclusion as to the operative technic. After exposure of the cord at the selected level, the cord must be rotated by grasping a denticulate ligament with mosquito forceps. This brings into view the anterolateral area. To section the latter, according to prescribed measurements, a special instrument should be used. The instrument I recommend is one in which a blade, with a curve 2.5 mm. in diameter, is mounted on a carrier at an obtuse angle. With this the anterolateral column is encircled and a second sliding blade is advanced until the tissue intervening between the two blades is severed. This instrument as nearly as possible assures an accurate performance of the operation.

THE RELATION OF THE CEREBRAL HEMISPHERES TO ARTERIAL BLOOD PRESSURE AND BODY TEMPERATURE REGULATION

PRELIMINARY NOTE *

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CHICAGO

It has been pointed out elsewhere [1] that removal of the cerebral hemispheres and the optic thalamus destroys the mechanism for regulating body temperature and reduces the animal to a cold-blooded condition. This loss of ability to maintain a normal body temperature after extensive traumatic injury around the third ventricle is common to all warm-blooded animals from man to birds. Only one group of warm-blooded animals so far as the literature indicates will long survive such an operation. Dogs, cats and monkeys may be kept alive for several days, but in the dog, on which most work has been done, one week is the maximum recorded length of life. In birds, which are warm-blooded animals, with proper precautions the animal may be kept alive for several months.

In an attempt to analyze the factors that lead to this loss of ability to regulate body temperature, studies on the circulation of the blood in these animals have been started. A method was devised for measuring arterial pressure in the pigeon and determinations have been made before and after reduction of the animal to the cold-blooded condition.

In the normal adult pigeon under all stages of ether anesthesia the blood pressure in twenty-three birds ranged between the extreme limits of 98 to 180 mm. of mercury. The average value of these determinations was 121 mm. (Table 1).

Measurements of arterial pressure have been made on six pigeons reduced to the poikilothermous condition by removal of the thalamus and hemispheres. These have been made at time intervals of from three to thirty days after operation so as to exclude the possibility of dealing with the acute effects only. The body temperature of these birds was set at various levels by changing the temperature of the incubator in which they were kept. The results of this experiment are given in Table 2.

These results suggested that the loss of temperature regulation was the result of lowered arterial pressure. However, as a control on this

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* From the Hull Laboratory of Physiology, University of Chicago.

[1] Am. J. Physiol. **49**:271, 1919.

TABLE 1.—ARTERIAL PRESSURE OF NORMAL BIRDS *

126	114	108
106	100	122
142	124	136
138	124	124
116	118	122
160	102	98
110	119	110
150	110	

Average, 121.

* All readings are made in mm. of mercury.

TABLE 2.—ARTERIAL PRESSURE OF POIKILOTHERMOUS BIRDS

Days to Recover from Operation	Body Temperature	Blood Pressure
3	29 C.	78 mm.
5	28 C.	84 mm.
13	28 C.	95 mm.
5	36 C.	104 mm.
15	29 C.	93 mm.
29	41 C.	85 mm.
3	31 C.	80 mm.

TABLE 3.—ARTERIAL PRESSURE OF DECEREBRATE BIRDS; BODY TEMPERATURE NORMAL

Normal Pressure Before Operation	Days to Recover from Operation	Blood Pressure After Operation
128 mm.	8	105 mm.
156 mm.	11	97 mm.
108 mm.	3	93 mm.
98 mm.	16	96 mm.
134 mm.	3	98 mm.
142 mm.	5	122 mm.
124 mm.	5	103 mm.
...	75	105 mm.
...	21	96 mm.
...	6	87 mm.
...	11	91 mm.

conclusion, blood pressure readings were made on birds from which the hemispheres only had been removed, the thalamus being left intact, and which therefore had normal body temperature. It was found that loss of the hemispheres also led to a similar fall in arterial pressure, though not quite so extensive (Table 3). In different cases this ranged from 10 to 20 per cent. It has long been known that in acute experiments removal of the hemispheres leads to a slight fall in arterial pressure, but as is evident from the time figures given, this fall is more than a mere temporary shock effect, and may persist as long as two and one-half months after decerebration; and this occurs in animals in which we have been accustomed to think the shock effects are at a minimum.

A comparison of the arterial pressure in the two sets of operated birds, both with hemispheres removed, the one with the ability to regulate body temperature, and the other with temperature regulation impaired, indicates a greater degree of depression in the latter series. However, the difference is so slight that it suggests that the lowering of body temperature must be due to some other factor than that of circulatory disturbance alone. I suspect that the disturbance in body temperature is due not only to the vascular disturbance, but also to lessened heat production, in part if not wholly due to injury of the subcortical motor centers for the skeletal muscles.

In concluding, it may be added that the above described effects are inconstant in localized lesions of either the hemispheres or thalamus. Hence the effects described are, in this report, confined to those following total extirpation. Certain effects of lesser lesions will be described in the full report to be given later.

SUMMARY

Removal of the cerebral hemispheres in the pigeon, leaving the thalamus intact and body temperature normal, leads to a constant fall in arterial pressure of from 10 to 20 per cent. This fall comes on immediately and persists for as long as seventy-five days after the operation.

Removal of the hemispheres and thalamus leads to a loss of temperature regulation and usually to a slightly greater fall in arterial pressure than does loss of the hemispheres alone.

The poikilothermous condition that follows deep lesions of the brain stem is not due to changes in the arterial blood pressure alone.

ABSENCE OF LOBUS OLFACTORIUS AND SCLEROSIS OF CORNU AMMONIS *

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Cases showing absence of the olfactory lobe are rare. In 1914 Weidenreich[1] reported one case and quoted nine others from the literature on this subject. Kundrat,[2] in his monograph, "Arhinencephalie," describes brains which have no olfactory lobe, but are combined with other brain anomalies. Besides these two reports I was able to find one case reported by Valenti.[3] But according to Weidenreich all reports which he quoted lack detailed information, especially in that they do not discuss the area of the brain supposedly connected with the olfactory nerve and therefore called "rhinencephalon." Valenti's report contains no statement about this area.

In Weidenreich's case there was entire absence of the bulbus and tractus olfactorius in both hemispheres. We assume that the rhinencephalon governs the sense of smell as found by Broca and Zuckerkandl in their studies in comparative anatomy, and by Retzius embryologically. If this is true also in man, we should be able to see some change in the parts regarded as the olfactory center in brains that have no olfactory lobe. Weidenreich investigated carefully his case that dealt with the bilateral absence of the bulbus and tractus olfactorius, absence (right) and reduction (left) of the tuber olfactorius. Except for these defects, no parts of the brain regarded as the rhinencephalon showed any marked change which denotes atrophy; but he was not able to study his case histologically because of the unsuitable previous treatment of the material for this purpose. He concludes the question of the olfactory center with the sentence: "Auch die Lösung diesser Frage bleibt also aufgespart, bis der Zufall eine neue Beobachtung des Defectes bringt und zugleich die Möglichkeit einer sofortigen Untersuchung am frischen und entsprechenden fixirten Material."

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* From the Pathological Laboratory of the Massachusetts Commission on Mental Diseases, 74 Fenwood Road, Boston, Mass., U. S. A.

[1] Weidenreich: Ueber partiellen Riechlappendefekt und Eunuchoidismus beim Menschen, Ztschr. f. Morphol. u. Anthrop. **18**:157, 1914.

[2] Kundrat; Arhinencephalie, 1882, abstr. in Schwalbe; Die Missbildungen des Kopfes, 1913.

[3] Valenti: Un cas d'absence unilatérale de l'appareil olfactif, Arch. ital. de biol. **64**:457, 1911.

It is many years since Broca and Zuckerkandl announced that the gyrus fornicatus must be regarded as the cortical olfactory area (it must be understood that under the name gyrus fornicatus or rhinencephalon are included gyrus cinguli and gyrus hippocampi with cornu ammonis). Although I am inclined to believe that certain portions of the rhinencephalon govern the smell sense, the part that subserves this function is still unknown, especially in man.

The authors quoted are arranged according to the year of publication.

Examination of animals has convinced Ferrier (cited in Bechterew's paper) that the olfactory center is located at the lower extremity of the temporal lobe. Munk (also cited by Bechterew) seeks it in the neighborhood of the cornu ammonis.

Onodi[4] concludes, after investigation of the hitherto published clinicopathologic reports concerning the sense of smell, that the olfactory center might be traced into the gyrus hippocampi and the uncus.

Ramón y Cajal,[5] from a histological study of the brains of several animals as well as that of man, has reached the conclusion that the focus spheno-occipitalis, the subiculum, the focus pra-esubicularis and the cornu ammonis seem to have no direct connection with the olfactory nerve fibers, and that they probably represent the tertiary olfactory center.

Campbell,[6] chiefly from histologic study of the human brain, recognizes the cross relation between the lobus pyriformis (frontal pear-shaped part of the gyrus hippocampi) and the cornu ammonis, but he doubts whether Ramón y Cajal is right in regarding the latter as a tertiary olfactory center. One of his conclusions is "Histology supports comparative anatomy in suggesting that in the human brain the lobus pyriformis must be regarded as the principal cortical center, although not the sole one governing the olfactory sense."

Broadmann[7] distinguishes the gyrus hippocampi from the surrounding areas on the ground of the histologic construction and names it "area ento-rhininalis."

Bechterew[8] concludes, from the results of experimental investigation on sixteen dogs, that an olfactory center is located apparently in the lobus pyriformis, and that this must be regarded as the perception center for the sense of smell, while perhaps the neighboring region (subiculum cornu ammonis, Bechterew) may have the function of its conception.

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[4] Onodi: Die Rindencentren des Geruches und der Stimmbildung, Arch. f. Laryngol. u. Rhinol. **14**:74 1903.

[5] Ramón y Cajal: Studien über die Hirnrinde des Menschen, No. 4, 1903.

[6] Campbell: Histological Studies on the Localization of Cerebral Function, New York, G. P. Putnam's Sons, 1905.

[7] Brodman: Vergleichende Lokalisationslehre der Grosshirnrinde in ihren Prinzipien dargestellt auf Grund des zellenbanes, Leipzig, 1909. Brodmann: Feinere Anatomie des Grosshirns in Lawandowsky: Handbuch der Neurologie, Vol 1.

[8] Bechterew: Die Funktionen der Nervencentra, 1911.

Edinger[9] seems to approve the cornu ammonis for this center chiefly from the comparative anatomic standpoint.

As I have mentioned above, our knowledge of the olfactory center is limited. Campbell declared after covering the histologic evidence:

> Above all things we seem to lack the knowledge which in the case of some other cortical regions has proved so valuable. I refer to that derived from studies in pathological history; for instance, we have much to learn of the cortical changes attending uncomplicated atrophy or lesion of the olfactory bulb and peduncle, and have virtually no conception as to what subdivisions such changes would be distributed over.

Fortunately, through the generosity of Dr. Southard, I was permitted to investigate a brain that had no olfactory tracts. I will give the macroscopic and microscopic findings of this rare and interesting case, describing particularly those parts of the brain which have been suggested as having intimate connection with the olfactory sense. I shall endeavor to throw some light on the question of the olfactory center which seems to be so obscure.

REPORT OF A CASE

History.—F. S. H. 228, S. B. I. 1916. 87 (from the record of the Foxborough [Mass.] State Hospital), R. R., a man, aged 45, had epileptic dementia, the probable cause of which was heredity. The psychosis had been of gradual onset and of over fourteen years' duration, and had been characterized by periodic attacks of an epileptiform nature during which the patient lost consciousness, had generalized clonic convulsive movements, bit his lips and tongue and had loss of sphincter control. Following these attacks the patient was weak, befogged and depressed. He also had periods of depression and at times of excitement, auditory and visual hallucinations, with delusions of persecution. His judgment and emotional fields were markedly impaired, and there was general mental deterioration with no insight. He was admitted to the hospital April 17, 1907, and died July 13, 1916. Necropsy was performed by Dr. M. M. Canavan, eleven hours after death.

Anatomic Diagnosis.—This included brown atrophy of the heart muscle; ascites; calcified lymph node in mesentery; bladder distended; chronic perisplenitis; sclerosis of the mammary arteries; hydrothorax; hydropericardium; sclerosis coronaris; tricuspid and mitral stenosis; chronic fibrous endocarditis, mitral; chronic fibrous endocarditis, aortic; acute vegetative endocarditis, aortic; possible bronchopneumonia; pulmonary infarcts; accessory spleen; uric acid deposits, kidney (?); pigmentation, fibrotic spots; focal opacities, pia; absence of lobus olfactorius; soft cerebellum.

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[9] Edigen: Vorlesungen über den Bau der nervosen Zentralorgane, 1911.

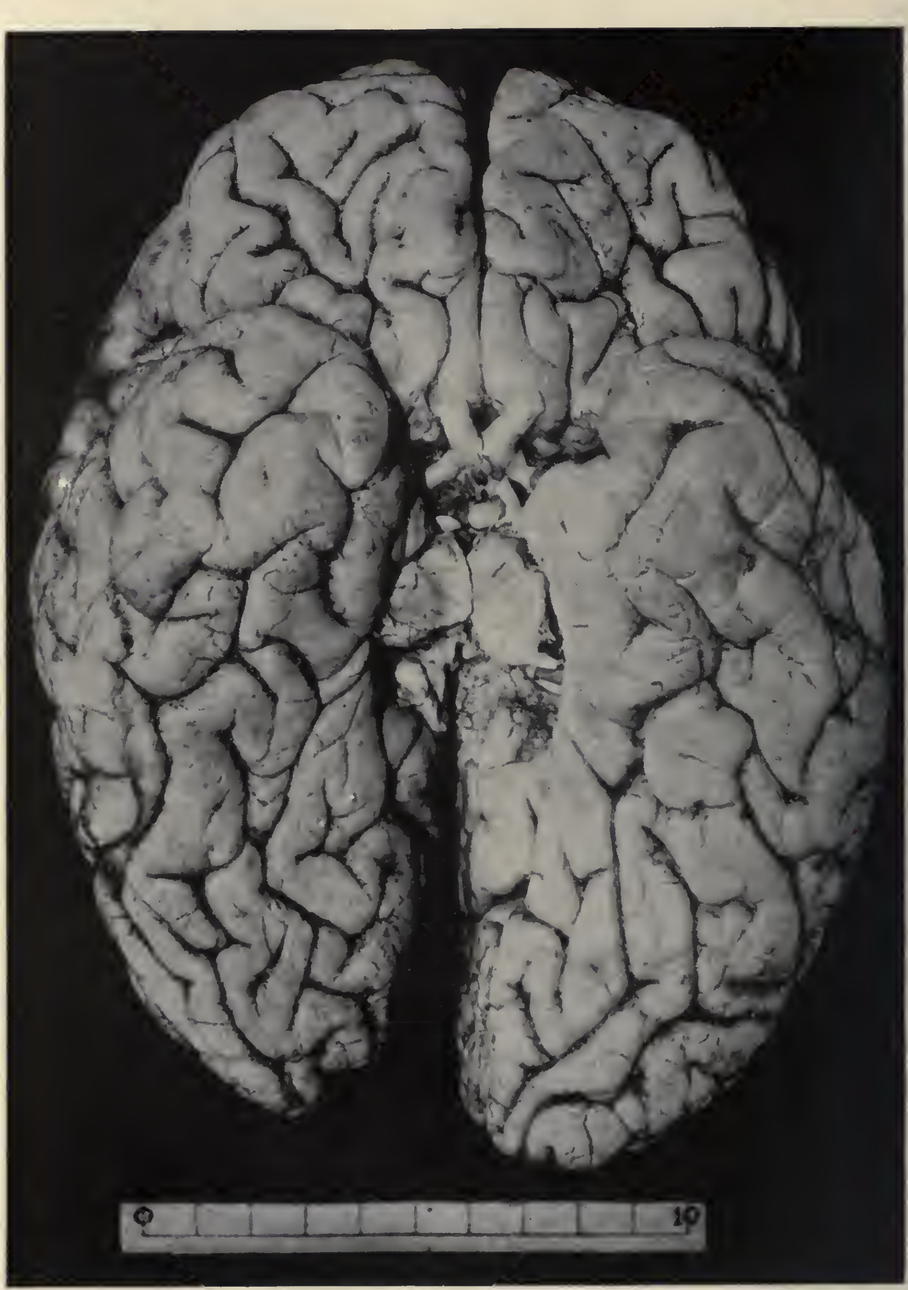

Fig. 1.—The base of the brain entirely bereft of bulbus and tractus olfactorius; rudimentary development of trigonum olfactorium and sulcus olfactorius are noticeable.

MACROSCOPIC STUDY OF THE BRAIN

In general form and size the brain was about normal. The pia mater showed focal opacities in the frontal and motor regions, and was nonadherent. The gyri in general were of normal richness and appearance and almost symmetrical. On the base of the brain the abscence of the bulbus and tractus olfactorius first attracted our attention (Fig. 7). There was slight sclerosis in the basal arteries. On careful stripping of the pia mater the findings in the rhinencephalon were: (The nomenclature in the following description has been adopted chiefly from Retzius.[10])

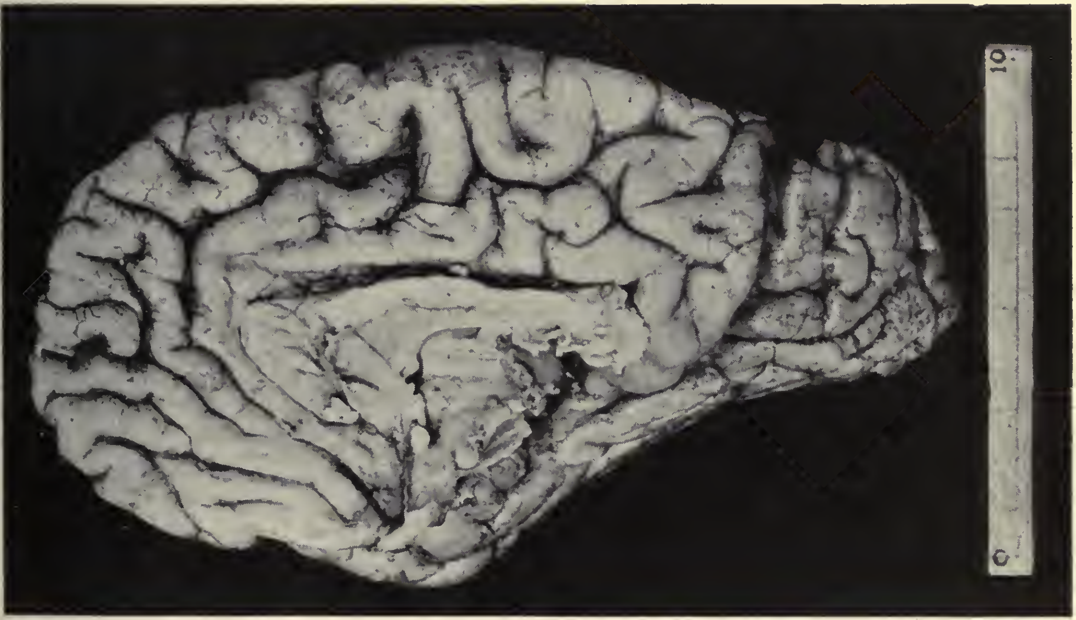

Fig. 2.—Mesial view of right hemisphere of the brain (see the description of macroscopic findings).

Right Hemisphere.—The weight of the right hemisphere after hardening in formaldehyd was 604 gm. The bulbus and tractus olfactorius were absent. Corresponding to the area of the trigonum olfactorium a flat and small pyramidal pointed process could be seen; both its sides and tip were free but the brain substance beneath adhered to its base. The tip of this process rested in a rudimentary sulcus olfactorius; no filia was to be seen. A rudimentary sulcus olfactorius ran forward from the top to the process described above for about 1.7 cm., and divided into two short transverse branches. As far as this sulcus extended a gyrus rectus could be distinguished but at the end of the sulcus the gyri were bounded by a short transverse gyrus. Gyrus tuber olfactorius, gyrus olfactorius medialis and lateralis were not definite. Along the posterior ends of the gyrus rectus and gyri frontales a narrow convolution, a continuance of the area parolfactorici on the mesial surface, ran transversely into the limen insulae and separated the surface connection between the frontal lobe and the anterior perforated substance. The space of the latter was narrow. No stria of olfactoria was to be seen. Sulcus parolfactorius anterior connected upward with the sulcus cinguli, and by the sulcus parolfactorius we could distinguish area parolfactoria and gyrus subcallosus, both pointing upward to gyrus cinguli (Fig. 2). The gyrus hippocampi was bounded anteriorly by incisura temporalis, above by the fissura rhinica in its anterior portion, and by sulcus collateralis on the posterior border; its length from the frontal tip to gyrus rhinencephalolingualis was about 4 cm. and seemed a little atrophied. From the inner sides of the hemisphere in which the crus cerebri was cut off could be seen clearly the whole length of the fascia dentate and the fimbria, and a part of the fornix through the dorsal surface of the gyrus hippocampi, with no pressing up of the thalamus. The gyrus semilunaris, sulcus semiannularis, gyrus ambiens and sulcus rhinica

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[10] Retzius: Das Menschenhirn, 1896.

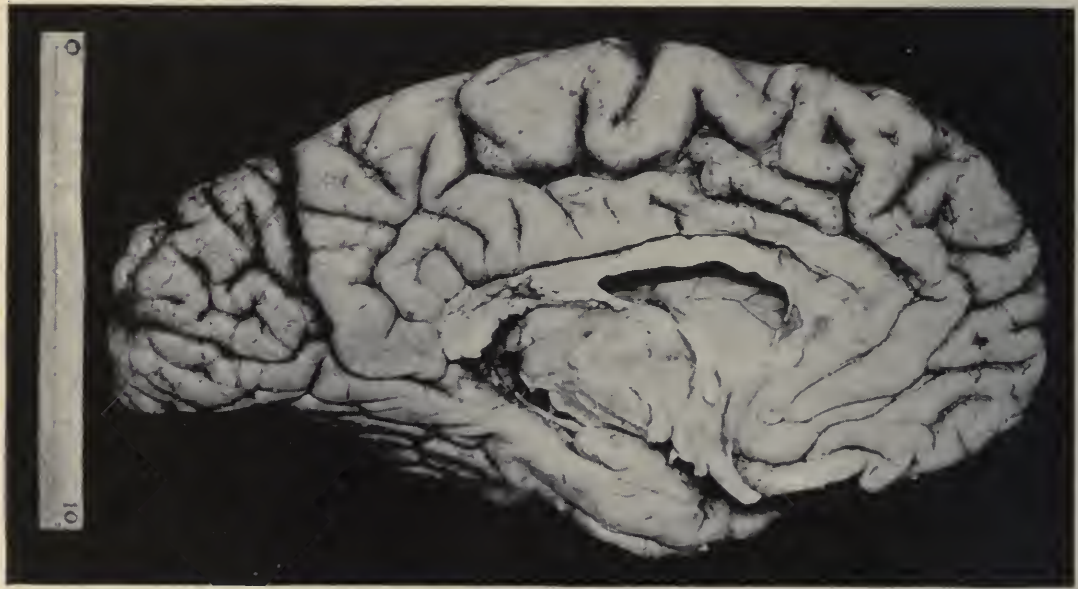

Fig. 3.—Mesial view of left hemisphere of the brain.

inferior, which Retzius noted especially in the gyrus hippocampi, were easily identified. The uncus was well developed and bent sharply against the gyrus hippocampi as usual. The gyrus fusiformis was separated from the former gyrus by the fissura rhinica in its anterior portion and posteriorly by sulcus collateralis, with the exception of a narrow communication, gyrus rhinencephalofusiformis, located between the former fissura and the latter sulcus.

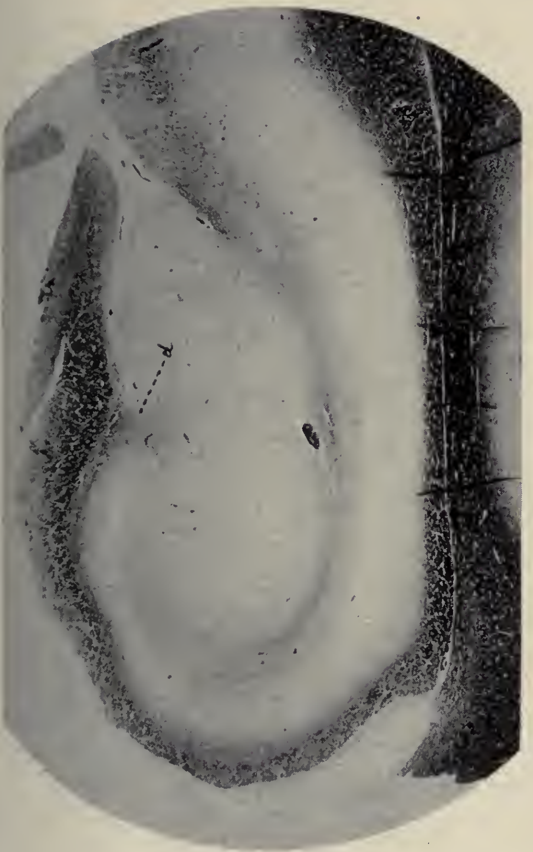

Figure 4

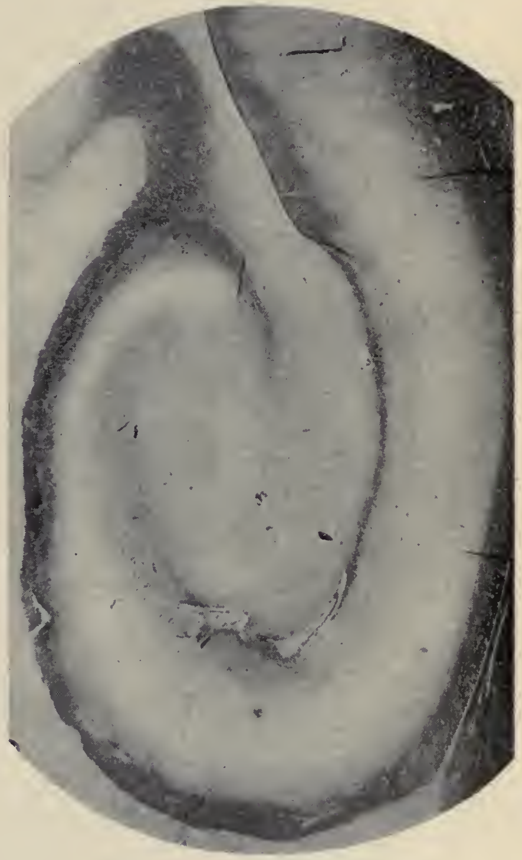

Figure 5

Fig. 4.—Section of the cornu ammonis (right), which is atrophied and the layer of gray matter remarkably narrowed. The deep branch of the alveus (*d*) which spreads into the terminal plate (into fascia dentate) is shortened and stains poorly (see also Figure 15). Compare this with Figure 5.

Fig. 5.—Section of about the same region of a normal cornu ammonis. Kulschitzky-Wolter's modification of Weigert's myelin sheath stain; thickness, 15 microns.

At its posterior extremity the gyrus hippocampi joined with the gyrus cinguli by gyrus rhinencephalolingualis, with gyrus cinguli and also with the precuneus by the isthmus cinguli as well as by precuneus anterior. The sulcus cinguli as the continuation of the sulcus parolfactorius anterior at its frontal end ran upward then backward around the genu corporis callosi and joined with the sulcus paracentralis, which separated lobulus paracentralis and precuneus. The gyrus cinguli was well developed and separated from the neighboring gyri by sulcus cinguli and subparietalis, from the corpus callosum by sulcus

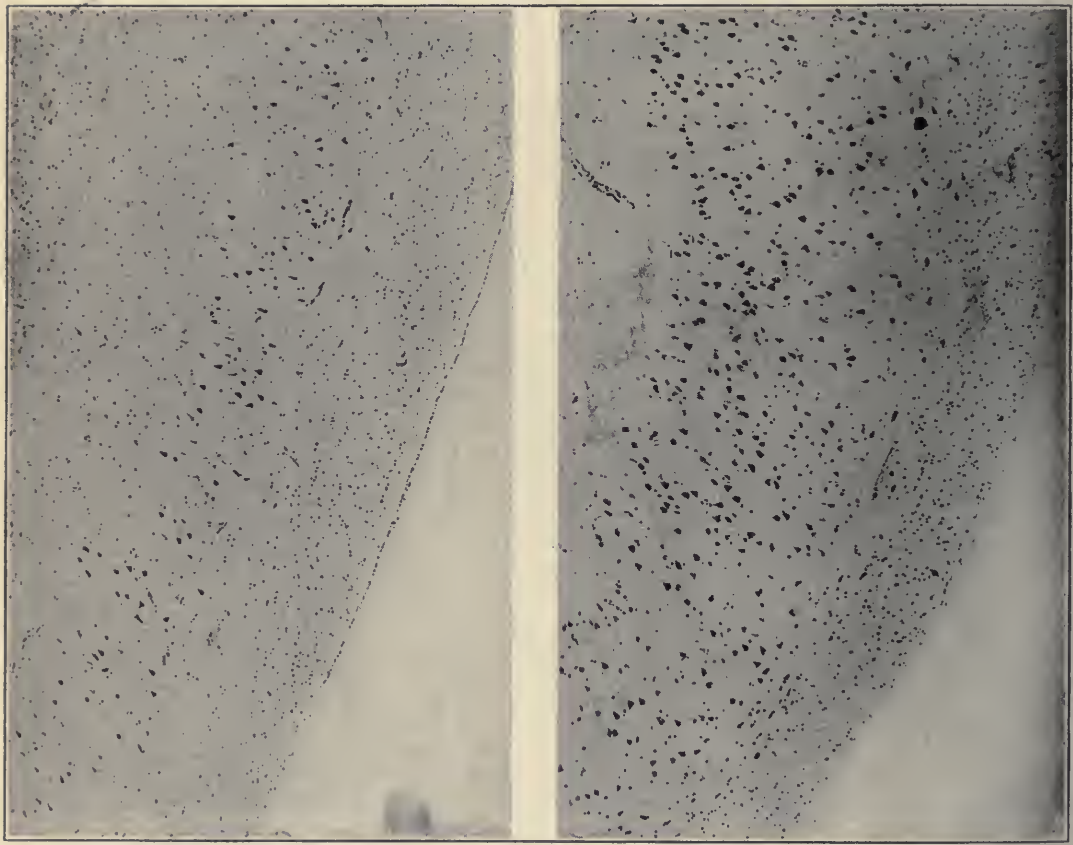

Figure 6

Figure 7

Fig. 6.—An area in the dorsal plate of a section of the cornu ammonis (left); cell layer is remarkably narrowed, pyramid cells are reduced in number and atrophied; thickened walls of blood vessels are also noticeable. Compare this with Fig. 7.

Fig. 7.—The same area as in Figure 6 of about the same region of a normal cornu ammonis. (Cresylviolet stain in paraffin section after formaldehyd and alcohol fixation; thickness 6 microns.)

corporis callosi. Its frontal end communicated with area parolfactoria and gyrus subcallosus; its posterior part was joined to the gyrus hippocampi. In the commisura anterior there was no notable change.

Left Hemisphere.—The weight of the left hemisphere after hardening in formaldehyd was 607 gm. The bulbus and tractus olfactorius were absent. In the place of the trigonum olfactorium there was a process of pyramidal form about the size of a kernel of corn, but it was not so flat as that of the right hemisphere; its tip was slightly free but the other parts adhered to the frontal lobe beneath. Gyrus tuberis olfactorii, gyrus olfactorius medialis and lateralis could not be differentiated; neither could the filia nor the stria olfactoria. The gyrus rectus could not be differentiated from the gyri frontales, and a sulcus olfactorius did not exist. The space of substantia perforata anterior was narrowed and separated from the orbital surface by a sulcus which ran from the mesial surface as the continuation of sulcus parolfactorius posterior. The gyrus hippocampi was 5 cm. long from its frontal tip to the gyrus rhinencephalolingualis and was bounded from the gyrus fusiformis by the sulcus collateralis. The fissura rhinica, gyrus semilunaris, sulcus semiannularis, gyrus ambiens and sulcus rhinica inferior could all be recognized. Generally the gyrus appeared to be a little atrophied, especially on the dorsal surface near the fissura hippocampi, where the convolution flattened and clear strands of the fascia dentata, fimbria and a part of fornix were to be seen from the inner sides of the hemisphere (Fig. 3). The sulcus cinguli, beginning as the continuation of sulcus parolfactorius anterior at its frontal end ran as usual almost parallel to the corpus callosum and combined posteriorly with the sulcus paracentralis. Gyrus cinguli, bounded superiorly by the sulcus cinguli and sulcus subparietalis, and below by the sulcus corporis callosi, was well developed and its posterior end reached to the isthmus rhinencephalolingualis as usual. The anterior end of the gyrus cinguli joined below to the area parolfactoria and gyrus subcallosus, which were as well developed as those of the right hemisphere.

HISTOLOGIC STUDY

Although there is no definite proof of the location of the olfactory center in the human brain, it is not difficult to assume that either the gyrus hippocampi or the cornu ammonis may govern the function. Therefore I made it my chief object in the microscopic study to examine these two regions. I took out the whole gyrus hippocampi with the cornu ammonis and the fascia dentata from both hemispheres. I investigated the fine construction and the lamination of nerve cells by the cresyl violet stain in paraffin sections, the arrangement of nerve fibers by Kulschitzky-Wolter's modification of Weigert's myelin-sheath stain in celloidin sections.

To obtain exact results I prepared as many serial sections as possible, and in order to avoid any misjudgment by comparison I investigated by the same methods the same regions of four other specimens which I had obtained from four normal looking hemispheres. I also examined other important areas of the brain.

Gyrus Hippocampi.—Cajal and Campbell have studied the normal histology of the gyrus hippocampi in detail. I state the findings in my case chiefly according to the topographical subdivisions by Ramón y Cajal, at the same time taking into consideration Campbell's description.

(*a*) The subiculum plexiform layer was about of normal thickness; deep in this layer islets composed of numerous minute triangular cells were seen as usual. Four or five of them were found in a section of the frontal area but much fewer in the posterior area. The pyramid and polymorphous cell layers were narrowed and many nerve cells, especially the pyramidal cells, were moderately shrunken with more or less affected nuclei. There was no change in the lamination and the cells were arranged in regular parallel rows as usual. Glia cells seemed to be increased.

(*b*) Praesubiculum: The tangential fibers in the plexiform layer ran irregularly; some were swollen and pear-like in appearance. The thickness of the gray matter beneath was reduced and many nerve cells, especially the pyramid cells, were moderately shrunken, but we were able to recognize the small pyramid and fusiform cell layer, the deep plexiform layer, the medium sized and large pyramid cell layer and the fusiform and triangular cell layer, as described by Cajal.

(*c*) The Hippocampal Gyrus Proper: The tangential fibers in the plexiform layer ran irregularly and were much fewer than normal. In the layer containing a cluster of polymorphous cells, the clusters of large polymorphous cells which characterize this area of the gyrus were to be found at the lobulus pyriformis, less toward the posterior end, but their protoplasm as well as their nuclei were markedly shrunken. The deep plexiform layer or medium sized pyramid cell layer, the large pyramid cell layer and the fusiform and triangular cell layer could all be differentiated, but many nerve cells, especially the pyramid cells, were considerably shrunken. As a whole, the cortex was narrowed. In Weigert's slides, there was no notable change in the arrangement of the radial fibers in the gray matter, but the interradial fibers among the former were few in comparison with those in brains having olfactory tract intact.

Cornu Ammonis and Fascia Dentata.—The histology of these organs in the human brain has been studied carefully by Kölliker,[11]

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[11] Kölliker: Handbuch der Gewebslehre, 6 te Aufl. 1896.

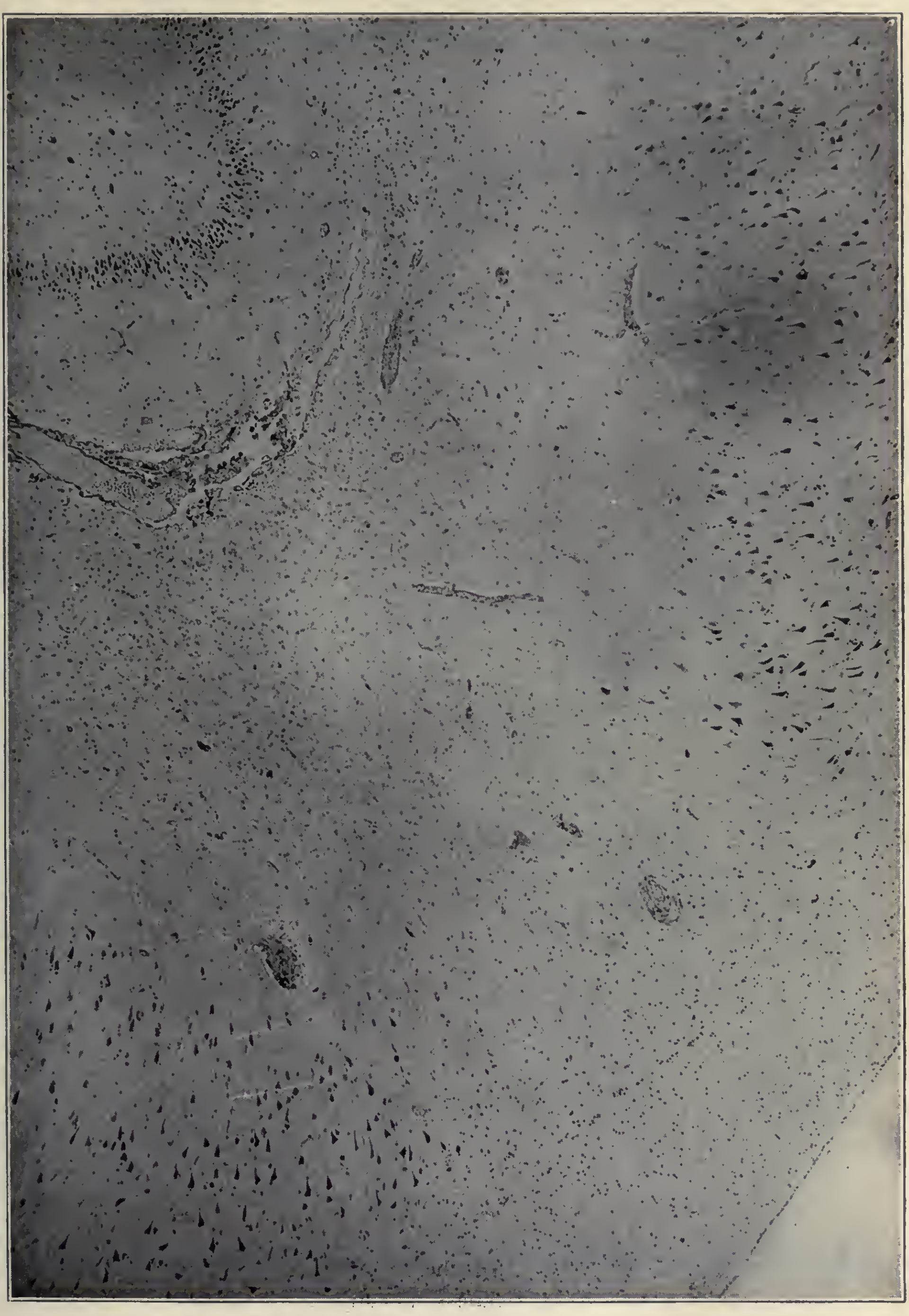

Fig. 8.—A gross gap of cell layer in the cornu ammonis (left). The pyramid cells have entirely disappeared in this lesion and it is filled up by neuroglia. Atrophy of pyramid cells is also noticeable. Formalin and alcohol fixation; cresyl violet stain; thickness 6 microns.

and Doinikow,[12] in his study of camparative anatomy, agrees with Kölliker.

(*a*) The Cornu Ammonis: The small size and the marked narrowing of the gray matter were noticeable at the first glance (Fig. 4).

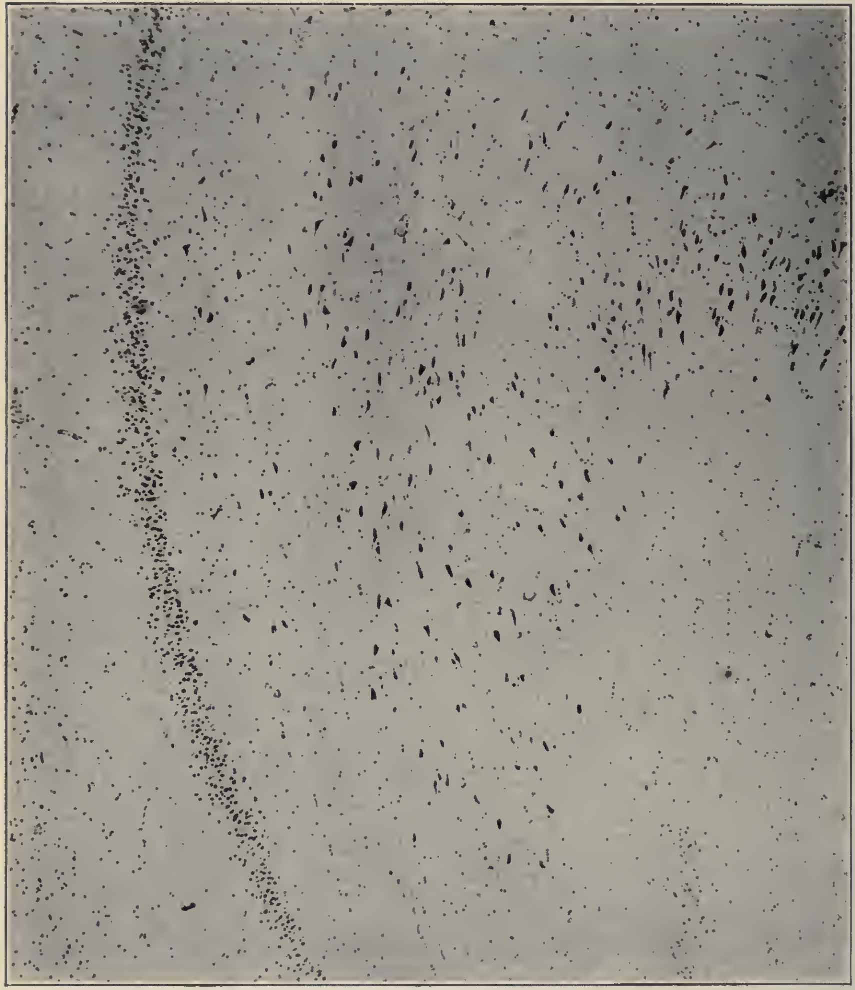

Fig. 9.—Alteration of nerve cells in the terminal plate of the cornu ammonis (left); they are reduced in number and markedly atrophied. Fine construction of these nerve cells can be seen in Figure 10. Compare with Figure 11.

The microscopic investigation of the sections disclosed: the stratum zonale was about normal in thickness; the superficial tangential, middle longitudinal and deep tangential fibers could be differentiated. The glia cells were increased and the moderate thickening of the wall of the blood vessels was visible. The cell layer, stratum cellularum pyramidarium and stratum oriens were markedly narrowed as we recognized with the naked eye, and it was a striking fact that the pyramid cells which characterized this layer by their large size as well as by their number were not only much reduced and atrophied (Fig. 6), but in some positions they had entirely disappeared and these gaps of the cell layer were filled with neuroglia (Fig. 8). Many of the pyramid cells in this layer were also markedly shrunken; their Nissl's bodies could not be differentiated and their nuclei had disappeared. Such changes of the nerve cells could be seen through the whole cornu ammonis; generally they were more noticeable in its dorsal plate than in its ventral and they were most striking in the terminal plate, as seen in Figures 9 and 10. In the area near the gaps described the change of the pyramid cells was also remarkable and in these situations the several stages of the cell alterations could be seen; some of them maintained their cell bodies only as a thread-like shell, while others kept their pyramid form with more or less affected protoplasm and nuclei. The favorite locations of the gap seemed to be the transitional regions of each plate into the other, and as far as I examined I found two spaces in the frontal part of the left cornu ammonis and individual spaces in the frontal and in the middle part of the other.

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[12] Doinikow: Beitrag zur vergleichenden Histologie des Ammonshorns, Jour. f. Psychiat. u. Neurol. **13**:166, 1908.

In Weigert's stain not only could the narrowing of the layer of gray matter be seen clearly, but places in which the gray matter had almost disappeared and nerve fibers were pressed together as if they connected the stratum zonale with the alveus crosswise could be seen (Figs. 4 and 13).

The Alveus: This was about normal in thickness or thicker than normal along the dorsal plate; it divided into two branches as usual — the superficial branch went into the fimbria without notable alteration, but the nerve fibers of the deep branch which spread among the nerve cells in the terminal plate of the cornu amonis were few and fine in comparison with those of the normal sections (Figs. 15 and 16).

(*b*). The Fascia Dentata and the Fimbria: In the stratum zonale nothing important was found.

Stratum Granulosum and Stratum Polymorpheus: The form and fine construction of each cell, as well as the number in a row, were about normal except in one area in which the ventral portions approached the surface; in this lesion many of the cells were moderately shrunken with alteration in their nuclei.

The Fimbria: There was no noticeable change in the fimbria, except that there were many blood vessels with thickened walls among the bundles of nerve fibers.

Other areas of the brain investigated were: The gyrus cinguli, cyrus temporalis superior, gyrus frontalis superior, gyrus centralis anterior, gyrus centralis posterior and gyrus occipitalis superior.

Speaking generally, the changes which I recognized in these areas, so far as I examined them, were the moderate thickening of the plexiform layer, more or less shrinkage of nerve cells in the gray matter and light thickening of the wall of the blood vessels.

SUMMARY

There were bilateral absence of the bulbus and tractus olfactorius, rudimentary development of the trigonum olfactorium in both hemispheres, absence (left) and partial development (right) of the sulcus olfactorius, nondevelopment of the gyrus olfactorius medialis and lateralis as well as of the gyrus tuberi olfactori in both sides, absence of the stria olfactoria and some atrophy of the gyrus hippocampi in both hemispheres. The other areas, which are regarded as the rhinencephalon, showed no notable alteration.

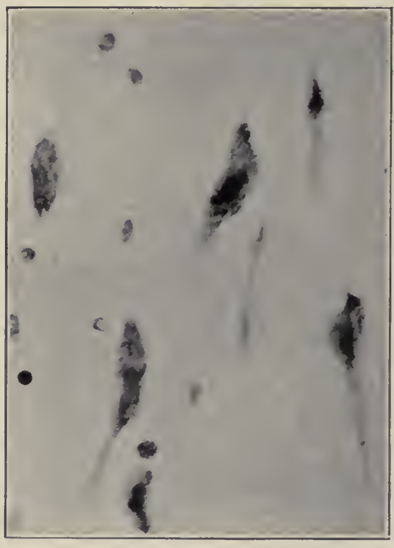

Fig. 10.—Alteration of nerve cells in the terminal plate of the cornu ammonis (left); they are reduced in number and markedly atrophied. Fine construction of these nerve cells can be seen in Figure 10. Compare with Figure 12.

Comparing this case with that of Weidenreich's, I find that there is no great difference between the two cases except in the incomplete development of the sulcus olfactorius and the atrophy of the gyrus hippocampi in my case.

As already mentioned, Weidenreich is of the opinion that, although the question of the olfactory center has not been solved by his case report, because no macroscopic change was to be seen in the rhinencephalon, perhaps the olfactory nerves connected the center in some irregular way, as the patient seemed to have had some sense of smell during life.

In my case, the absence of the gyrus olfactorius medialis and lateralis as well as of the gyrus tuberis olfactorii and the stria olfactoria—which were also lacking in Weidenreich's case—would not be of importance in regard to the question of the olfactory center, because the development of these gyri and stria depends directly on that of the bulbus and tractus olfactorius.

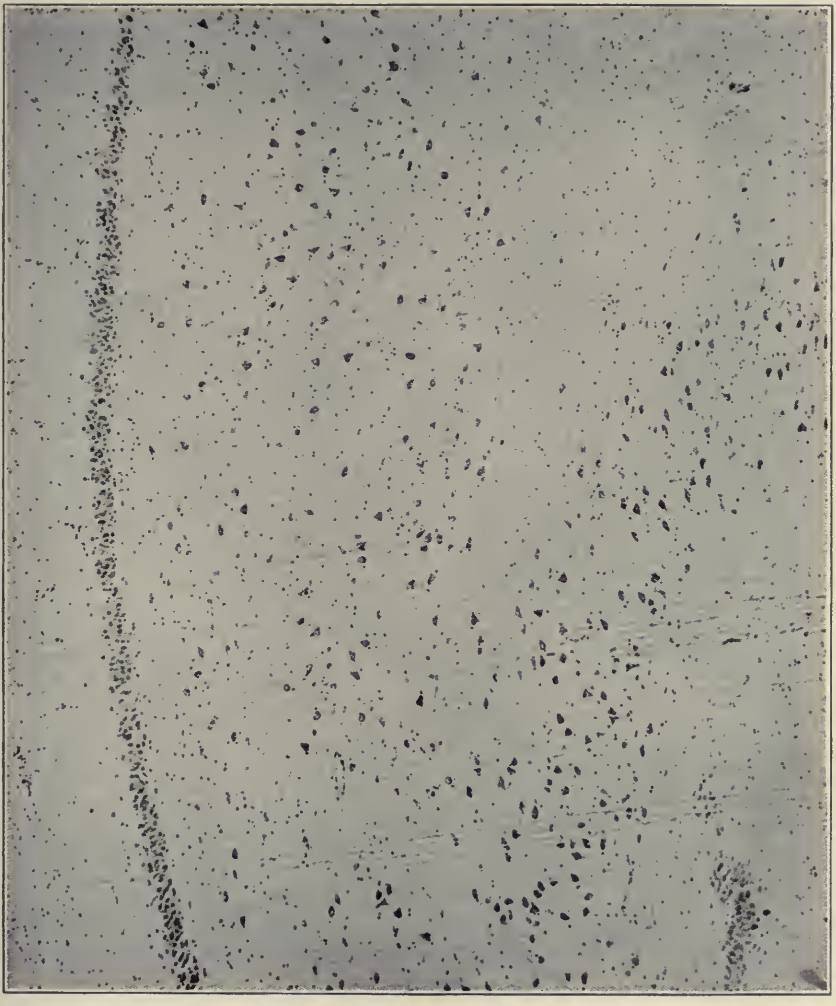

Fig. 11.—Nerve cells in the terminal plate of a normal cornu ammonis. Formaldehyd alcohol fixation; cresyl violet stain; thickness 6 microns.

The flattened gyrus hippocampi, which I have described, attracted my attention in this case, and it would be interesting to know whether this atrophy in the rhinencephalon is to be regarded as the result of the absence of the lobus olfactorius. It is to be regretted that I could not get any positive information about the sense of smell of the patient during life. Such an important question could not be solved by the macroscopic investigation alone, and therefore I discuss it later in reference to the microscopic findings.

Concerning the origin of the defect of the olfactory lobe, according to the description by Ernst,[13] arachinencephaly, a brain without an olfactory lobe, may develop when some compression acts on the head fold of the embryo. Schwalbe [14] states that the theory of compression as the cause of arhinencephaly and cyclopy has been shaken and the opinion that it is due to some embryonal defect is gaining more general acceptance.

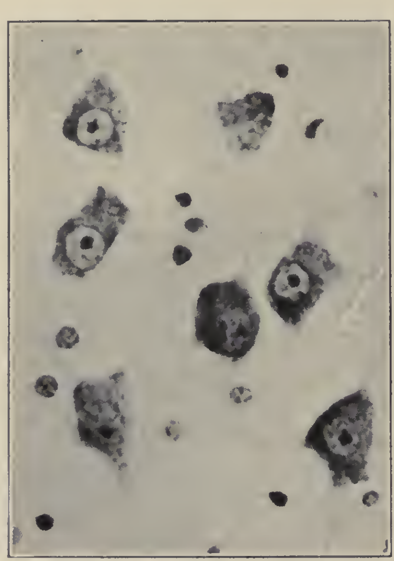

Fig. 12.—Finer details of these nerve cells shown in Figure 11. Formaldehyd alcohol fixation; cresyl violet stain; thickness 6 microns.

Weidenreich is of the opinion that in his case the olfactory nerve could not combine with the olfactory lobe as usual, and consequently the latter was deserted to atrophy. The full development of the sulcus olfactorius in his case shows that the olfactory lobe might have been developing for some time. He seeks the origin of the anomaly in some alteration of the embryonal connective tissue.

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[13] Ernst: Missbildungen des Nervensystem. Schwalbe: Morphologie der Missbildungen des Menschen und der Tiere, Teil III, Lief. 2, 1910.

[14] Schwalbe und Josephy: Die Missbildungen des Kopfes. Schwalbe: Morphologie der Missbildungen und der Tiere, Tiel III, Lief. 11, 1913.

In the present case the sulcus olfactorius did not develop so fully but, in accord with Weidenreich's, the main anomaly has been limited to the lobus olfactorius with no remarkable change in the neighboring

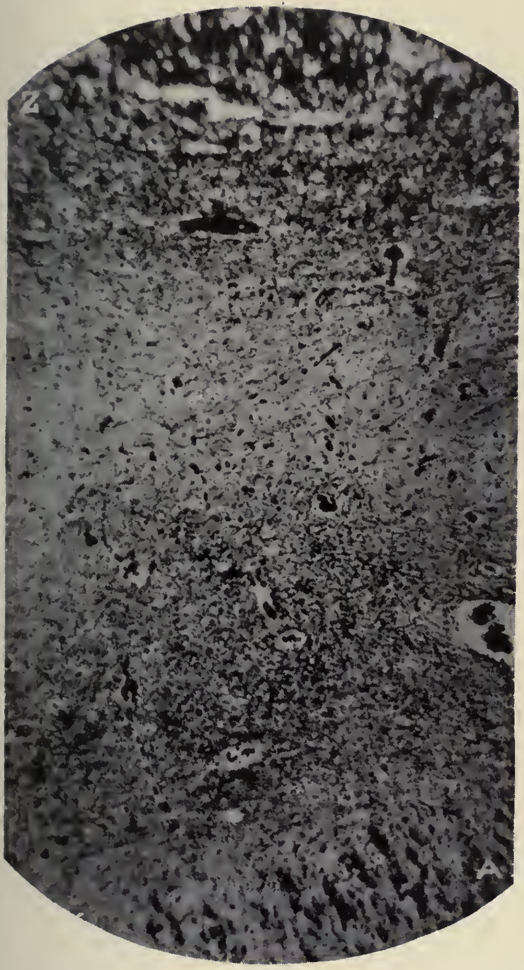

Figure 13

Figure 14

Fig. 13.—An area in the dorsal plate of the cornu ammonis (right); in this lesion the gray matter has almost disappeared and nerve fibers in the narrowed layer of gray matter are pressed together between stratum zonale (*Z*) and alveus (*A*). Compare this with Figure 14

Fig. 14.—The same area of about the same region of a normal cornu ammonis as in Figure 13 (Z — stratum zonale). Kulschitzky-Wolter's modification of Weigert's myelin sheath stain; thickness 15 microns.

areas of the forebrain. This is not to be explained by the compression theory, for if so, it must have been circumscribed as well as effective, and at the same time it must have acted on both hemispheres; but such a compression is not to be considered. I would not hesitate to believe that the defect of the lobus olfactorius might have had its origin in some defect in the embryonal tissue.

In the microscopic investigation the changes were found in the cornu ammonis. There were marked reduction in the gray matter, remarkable atrophy of pyramid cells and disappearance of the cell layer in some portions, fewer nerve fibers in the deep branch of the alveus, more or less increase of glia cells and thickenings in the walls of the blood vessels.

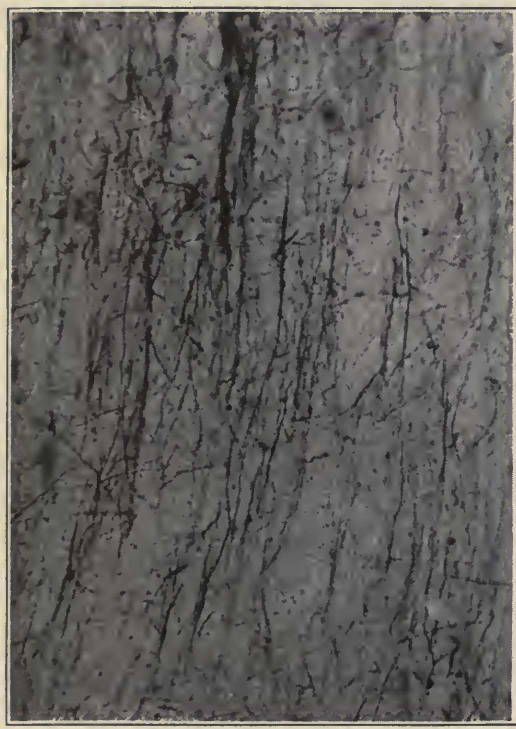

Figure 15

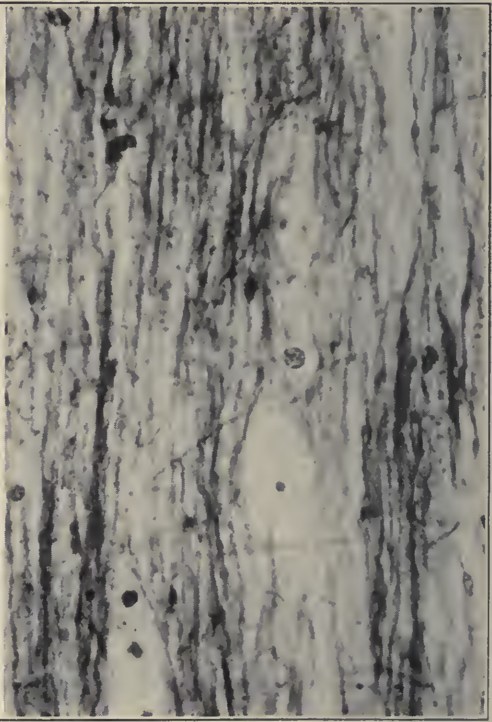

Figure 16

Fig. 15.—Fine construction of nerve fibers in the deep branch of the alveus (see also Fig. 4). The area presents itself just before the deep branch spreads among nerve cells in the terminal plate of the cornu ammonis (right). The nerve fibers are reduced in number and atrophied. Compare this with Figure 16.

Fig. 16.—Nerve fibers in the same area of about the same region of a normal cornu ammonis as in Figure 15. Kulschitzky-Wolter's modification of Weigert's myelin sheath stain; thickness 15 microns.

I believe that the change of the nerve cell denoted atrophy and not aplasia because the alteration was not the same through the whole cornu ammonis and, moreover, several stages of the change in the cells were seen.

Next to the cornu ammonis, the change in the gyrus hippocampi was most noticeable. The changes in this region were: less tangential fibers in the plexiform layer, marked atrophy of the superficial large polymorphic cells, moderate shrinkage of the pyramid cells and loss of the interradial nerve fibers.

It is interesting that to some extent these findings in the gyrus hippocampi accord with the result of the macroscopic investigation, which showed some atrophy of this gyrus. Moreover, it is extremely interesting to have found the most marked change in the cornu ammonis in my case, while the location of the olfactory center is still a question, and some authors, notably Ramon y Cajal, are suggesting the cornu ammonis as the terminal station of this sense.

According to His, quoted in Weidenreich's paper, the olfactory nerve develops separately from its central part and the connection with the brain is accomplished in a comparatively later stage. Therefore, it would not be unreasonable to expect some secondary change in the olfactory center, although it might have developed as usual. In this case in which there were no bulbus and tractus olfactorius, the cornu ammonis showed the most marked alteration in the so-called rhinencephalon.

Besides the anomaly of the olfactory lobe are there any other possible causes for this change to be considered?

Arteriosclerosis may be taken into consideration, as the thickening of the walls of the blood vessels was noted in the cornu ammonis; but the patient did not have marked arteriosclerosis and the basal arteries showed only a slight degree of the hardening. Hence an intense alteration in nervous tissue could not be explained by arteriosclerosis; moreover, the change in the nervous tissue was almost limited to the cornu ammonis.

Certain intoxications may be considered, but in the clinical history there is no evidence of any kind of intoxication as a probable cause of such a change in the nervous tissue. General intoxication would not cause the most marked change in the cornu ammonis; hence this cause may also be excluded. May I then conclude that I have eliminated doubt regarding the olfactory center, and that the cornu ammonis must be regarded as the terminal olfactory center?

Unfortunately, the patient suffered from epilepsy of long duration, as briefly described in the history. Since the sclerosis of the cornu ammonis in epilepsy was called to attention by Meynert, Sommer,[15] Bratz,[16] Arzheimer,[17] Worcester [18] and Southard [19] by histologic studies on this subject, I shall not give the results of the investigations of each one of these authors. It seems to be true that in several cases of epilepsy discussed by these physicians, some alterations — especially atrophy of pyramid cells, increase of glia cells and sclerosis of blood vessels were found in the cornu ammonis. This complicates the value of similar findings in the present case, as the defect of the lobus olfactorius cannot be excluded as a possible cause of these changes in the cornu ammonis. A discussion as to which of the two possible causes is more reasonable or was more effective in this case should be avoided, as any conclusion in this discussion appears to be only an assumption.

Although it is regrettable that this case could not bring out the soundest evidence necessary to solve the question of the olfactory center, I believe such a case and such an alteration in the cornu ammonis is a matter of enough interest to be reported and may be a contribution to further investigation on this question.

In closing this report I wish to acknowledge my great indebtedness to Dr. E. E. Southard and Dr. M. M. Canavan, whose kind help enabled me to investigate the brain discussed. I also wish to thank Mr. Herbert W. Taylor, a photographer, who prepared the photographs.

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[15] Sommer: Erkrankungen des Ammonshorn als aetiolog. Moment der Epilepsie, Arch. f. Psychiat. u. Neurol. **10**: 1880.

[16] Bratz: Ammonshornbefunde bei Epileptikern, Arch. f. Psychiat. u. Neurol. **31**: 1899.

[17] Arzheimer: Anatomische Untersuchungen am drei und sechsig Epileptikern. Jahresversammelung des deutschen Vereins für Psychiatrie, 1907; abstr. in Neurolog. Zentralblatt., 1907.

[18] Worcester, W. L.: Sclerosis of the Cornu Ammonis in Epilepsy, Tr. Nerv. & Ment. Dis. **56**:229, 1897.

[19] Southard: On the Mechanism of Gliosis in Acquired Epilepsy, Am. J. Insanity **64**:607, 1907-1908.

THE ASSOCIATION OF NEUROPSYCHIATRIC CONDITIONS WITH INFLUENZA IN THE EPIDEMIC OF 1918

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In the section of neurology and psychiatry of the office of the Surgeon-General of the Army are filed statistical and clinical records of over 70,000 neuropsychiatric cases reported from the various camps and hospitals in the United States during the war. Of these records, 269 have reference to influenza, the disease having been present at some time during patient's army service. A careful consideration of the records, however, made it apparent that in only seventy-three cases could influenza be deemed a significant factor in the development of the neuropsychiatric condition reported. These have been made the basis of the following study.

Neuropsychiatric Conditions Associated with Influenza

Infective-exhaustive psychosis	32
Dementia praecox	7
Manic-depressive psychoses	4
Psychoneuroses	19
Constitutional psychopathic state	2
Mental deficiency	2
Paralysis of the facial nerve	2
Neuritis	2
Cerebral embolism	2
Hyperthyroidism	1
Total	73

It is certain that the cases set apart for this discussion do not include all cases in the army that presented neuropsychiatric manifestations. Toward the latter part of the epidemic a number of cases undoubtedly were not brought to the attention of the section of neurology and psychiatry, owing to a change in the method of disposition of reports. It is also probable that many influenza patients had nervous and mental complications while under the care of the general medical officers, these cases not having been brought to the notice of, or placed in charge of, neuropsychiatrists. In connection with the question of the actual number of such cases, the experience of Major Egbert Fell at the Walter Reed Hospital is apropos. Dr. Fell states that of approximately 2,500 cases of influenza treated at the hospital, only twenty developed psychoses, and only four of these were of a severe type outlasting the acute disease.

The series of cases under consideration were widely distributed in the United States, being reported from eight camps, ten general hospitals and two embarkation hospitals. The largest group, comprising thirty-two cases, includes those which may be designated as the infective-exhaustive-toxic type. There were nineteen cases of psychoneuroses, four cases of manic depressive psychosis, seven of dementia praecox, four of neuritis and seven miscellaneous. The majority of these soldiers were within the first draft age. Twenty-two saw foreign service; the remainder did not leave the United States.

The group of thirty-two cases of the infective-exhaustive-toxic type comprises those which commonly presented marked physical reactions, and which appeared most properly to be connected with an acute illness such as influenza. These cases were variously diagnosed, such terms as infective, toxic, somatic, exhaustive, infective-exhaustive and toxic-exhaustive being used. It was rather usual to find evidence of physical exhaustion, e. g., tremor, anemia, loss of weight and so on. The symptoms varied from a mild confusion of bewilderment to a marked delirium with agitation, hallucinations, amnesia, ideas of persecution of hypochondriacal nature and other evidences of a severe psychotic reaction. In ten cases there is definite mention of hallucinations. Both restless excitement and depression are recorded as being about equal in frequency. Ideas of self accusation with a strong sexual coloring were present. In one case, there was a marked tendency to fabricate. The usual disorientation accompanied frank delirium. Of the thirty-two cases, ten patients were reported as returned to duty, ten more as recovered, seven recommended for discharge from the service and three were transferred to general hospitals for further treatment. In two cases there was no record as to disposition.

In only one of the thirty-two cases, had there been a previous attack or any history of predisposition. This soldier, a second lieutenant, had had a nervous breakdown seven years before, necessitating residence in a sanatorium and characterized by depression during which he cried easily. During his second psychosis he was excited, talked to every one about his trouble and had the idea that his stomach and testicles were eaten away. He thought he was being poisoned and vomited constantly. He was discharged from the service. The diagnosis of "infective psychosis" might be questioned, and the diagnosis of manic depressive insanity might be considered.

Seven patients had tainted family histories consisting of insanity, epilepsy, meningitis, neurotic disposition; none of these appeared to be of special significance.

Of this group the following case is abstracted to illustrate simple confusion and bewilderment with final recovery.

REPORT OF CASES

Case 1.—*History.*—Private B., aged 31, single, white, an automobile mechanic, had one sister who was nervous and two paternal uncles who were alcoholic. Otherwise the family history was negative so far as ascertained. As a child he had measles, mumps and scarlet fever. He attended school from the age of 6 to 15, reaching the eighth grade. He was active in sports. As an automobile mechanic he worked steadily, earning $5 per day. He denied venereal disease, and was a moderate user of beer.

He was drafted May 1, 1918, and was sent to Camp Humphrey, May 23, 1918.

Present Illness.—About October 10, he was taken with influenza. He was in barracks until October 17, when he was admitted to the Base Hospital. On October 24 he was found wandering about the Officers' Ward at the Base Hospital. He seemed to be bewildered and was transferred to the neuropsychiatric ward for observation.

On admission he was in a poor state of nutrition. He was disoriented for time, but not for place or person and was fairly coherent and relevant but complained of being "mixed up in the head." No hallucinations were noted. He constantly ran to the office saying he had a ticket and wanted to catch the next train home. He appeared to be "demented," and it was almost impossible to keep him in bed. He had difficulty in understanding what was said to him.

He gradually improved and finally recovered. At first his memory for recent dates was impaired, but this returned and he was perfectly clear on Nov. 7, 1918. He was returned to duty.

The next case presents symptoms of confusion, restlessness, hallucinations and ideas of persecution with final recovery. There were decided signs of physical exhaustion. He had also an attack of measles.

Case 2.—*History.*—Private G., aged 24, single, white, a farmer, had a negative family history so far as could be ascertained. Little information was received about his early life. He had had jaundice and chickenpox. He was a moderate user of beer. He had attended college for one year.

Present Illness.—On Sept. 15, 1918, after being in the service about three months, he developed influenza. Prior to this he had symptoms of measles; he was restless, rather silly and confused. For a time he heard voices telling him he was to die and saw Germans digging a trench in which to bury him. He thought he was accused of being pro-German and a spy.

Physically, he was poorly nourished, anemic and under weight. The pupils were unequal, but reacted normally. The blood Wassermann test was negative.

He gradually improved and recovered by Jan. 17, 1919. He returned to duty.

The following case was characterized by delirium, hallucinations and amnesia.

Case 3.—*History.*—Private H., aged 29, married, of mixed race, a farmer, had a negative family history as far as it could be ascertained. The history of his early life was negative. He reached the sixth grade in school. He was married and in comfortable circumstances. He was drafted June 17, 1918, and sent overseas four months afterward, remaining three months and then returned to the United States.

Present Illness.—He contracted influenza on board ship on the trip over, being admitted to a base hospital on Sept. 24, 1918. He was then confused, disoriented, restless and had auditory hallucinations. He improved but remained dull, perplexed and worried for some time. Later he recalled the trip over and back but remembered little, if anything, of his psychosis. By March 5, 1918, he was considered well and he was sent home with an attendant.

One soldier showed a tendency to fabricate which persisted for a week.

Case 4.—*History.*—The family history of Private H., aged 21, single, white, farmer, could not be ascertained. Very little was known of his personal history. He was said to have reached the second grade in school and could read and write. He had always worked on a farm. He never used alcohol or drugs. He served in the army four months, remaining in the United States.

Present Illness.—He had influenza with bilateral bronchopneumonia. The psychosis continued three weeks. Influenza began Sept. 28, 1918. By Oct. 17, 1918, he had recovered from his bronchopneumonia but at that time phlebitis of the left saphenous vein was noted. On Oct. 30, 1918, his first mental symptoms were observed. He was disoriented but cooperated well. He told of having enlisted in the regular army a year before, but was unable to give the data. He stated also that he had been in France, had participated in a bayonet charge and fought for half a day, but he could not give the name of the battle. He said that he had landed in New York about two months before. This was all pure fabrication and he continued this prevarication for a week, after which his mental condition cleared up and he became apparently normal. He was finally discharged from the service.

There were several cases of dementia praecox, six reported of the hebephrenic type, one a simple form. All were white, and their ages ranged from 21 to 31 years. Three saw foreign service, the others remained in the United States, the length of service ranging from three months to over two years. Six were eventually recommended for discharge from the service, two being transferred to St. Elizabeth's Hospital, and one continued under treatment in another army hospital. All were recorded as in line of duty with one exception.

Several of these soldiers served from six months to over one year in the United States prior to going to France where influenza was contracted. From the length of service in this country and the fact that they were selected for overseas duty, probably undergoing two neuropsychiatric examinations, one at the beginning of their service and the other for overseas duty, it may be supposed that they showed few if any mental abnormalities, and that the psychosis as it later appeared in association with influenza was a new development. Other writers have commented on the onset of dementia praecox as an acute psychosis with or following influenza, and these cases would appear to confirm such a possibility. On the other hand, at least one of these soldiers gives the history of having "always been seclusive" and showing a lack of initiative. From this, it would also appear that there may be in the patient a background of peculiar makeup, on which as a basis the acute illness brings to light the psychotic condition.

In five of these cases auditory hallucinations were present. In several patients, ideas of persecution were reported, such as that food was poisoned; but these symptoms were evidently not so prominent as to cause the examining officers to favor a diagnosis of paranoid praecox. One case began with great excitement and psychomotor unrest followed by rather rapid deterioration, especially in the emotional field.

The following case illustrates the onset of dementia praecox, hebephrenic form, after some months of apparently efficient army service, following an attack of influenza while in France.

Case 5.—*History.*—Private F., aged 30, single, white, farmer, had a negative family history, so far as could be ascertained. Little information could be obtained about his early life. He had measles in 1910. He denied syphilis and alcoholism.

Present Illness.—After six months' service in the United States, he contracted influenza in France in October, 1918. He became seclusive and had auditory hallucinations, hearing voices of girls using indecent language and asking him to marry them. He asserted that people read his mind. He talked little. He was oriented for time and place. With the exception of an acne eruption on the face, his general physical condition appeared to be good.

He continued under observation for about six months, finally being discharged.

The following case illustrates the onset of dementia praecox after a longer period of army service both in the United States and in France.

Case 6.—*History.*—The family history of Private P., aged 25, single, of mixed race, a farmer, was negative, so far as could be ascertained. Little information as to early life could be obtained. He reached the eighth grade in school. He was drafted July 22, 1917, served one year and four months in the United States, and one year in France. He was a moderate drinker.

Present Illness.—He contracted influenza while in France in October, 1918, having severe chills and fever, a cough and pain in the left side of the chest. He talked incoherently and excitedly. There were marked psychomotor activity and visual hallucinations.

This mental state continued although his physical condition became normal. In December, 1918, he became quiet and apathetic and irrelevant in conversation, showing evidence of hallucinations and delusions. He said that God talked with him; that his food was poisoned; that his bones seemed to draw electricity from the lights in the building. He talked continuously to himself. Orientation and memory were defective. After four months of observation, he continued to be disturbed and resistive, apathetic, untidy and expressed the same ideas. He was transferred to St. Elizabeth's Hospital, Washington, D. C.

With no history of previous peculiarity aside from shut-in personality, the following officer developed dementia praecox after influenza and bronchopneumonia.

Case 7.—*History.*—Lieutenant G., aged 30, single, of mixed race, a dentist, had a negative family history so far as could be asscertained. He was of the "shut-in" type of personality. Little information about further details of personal history could be obtained. He denied syphilis, alcoholism and drug addiction.

Present Illness.—He had influenza followed by bronchopneumonia in December, 1918. He complained of feeling "nervous" and "fearful"; he evaded questions and resisted examination, and thought the latter was for the purpose of circumcising him.

He had periods of excitement when he became noisy and destructive. He was unstable emotionally, frequently crying without reason, and talked of being related to Jesus Christ. He said he was to marry a girl who would bear him a child who would be a second Jesus Christ. He worried over a real financial indebtedness, but memory was clouded for recent events in general.

At first he seemed to have increasing insight and to be improving. Later he became more tractable, laughing easily, showing marked religious tendencies and reading the Bible much of the time. He spoke of "raising thoroughbred horses" as his "natural vocation," and dentistry as his "artificial vocation." He had some idea that his food was poisoned.

He was under treatment in four army hospitals, going away without official leave while in the second and going to Washington to interest the President in moving the World's Peace Congress to the United States; he was apprehended in Washington. He seemed to be deteriorating as evidenced by carelessness in appearance. He thought his case was parallel to that of Joan of Arc.

Of the four cases of manic-depressive insanity, three were cases of depression. Three patients had seen considerable service, one nine months in the United States and five months in France, another six months in the United States and twelve months in France, the third seven months in the United States and eight months in France. One of these cases had been diagnosed in France as "hysteria." Another patient who had profound depression with suicidal threats, had had a previous attack three years before which lasted for six months. A third, a negro, attempted suicide by cutting his throat with a razor during convalescence. He recovered. The fourth patient showed excitement after three months of service in the United States, characterized by a mild exhileration with restlessness and talkativeness, with final recovery; he returned to duty after two and one-half months.

The patient in the following case showed symptoms of a mild manic attack, with eventual recovery. A history of syphilis and a few suggestive physical signs complicate the picture. There is no record of a Wassermann test of the blood or a spinal fluid examination.

CASE 8.—*History.* Private P., aged 26, married, English, a chauffeur, had a negative family history for nervous and mental diseases so far as could be ascertained. His father had tuberculosis. Little information as to personal history could be obtained. He reached the eighth grade in school. He had pertussis at the age of 8, measles at 10 and mumps at 15. He was a moderate drinker. He contracted syphilis about three years before.

Present Illness.—After three months' service in the army, he had an attack of influenza on Oct. 19, 1918, remaining in the hospital eight days. On November 8 he sprained his wrist and was in the hospital for about two months.

He was restless, mildly exhilerated and talkative. He spoke constantly of army experience and showed much egotism. Orientation and memory were undisturbed. Judgment was poor.

Physical Examination.—All deep reflexes were exaggerated. The right pupil was slightly irregular in outline. Reacton to light and accommodation was fairly good. He complained of failing vision.

Course.—He made good progress and was recommended for return to duty, but was transferred for demobilization Jan. 31, 1919.

The next patient not only had a previous attack of mental trouble before army service, but also had a psychoneurotic manifestation from which he recovered, prior to the psychosis following the attack of influenza. The latter was characterized by marked depression with eventual recovery.

Case 9.—*History.*—Private F., aged 22, single, American, a college student; his mother was said to be insane. Little information was available. As a child he had measles and whooping cough, and chickenpox when 18. When drafted he was in his second year in college.

Three years before enlistment, he had a "nervous breakdown" that lasted six months during which he was depressed, had difficulty in thinking and contemplated suicide. He enlisted in June, 1917, and went overseas in August of the same year. In October, 1917, he "broke down," having a jerking and twisting of the head. He returned to his company in December and was well until July, 1918.

Present Illness.—In July, 1918, he had influenza. Following the acute febrile condition, he became stupid, dazed, had difficulty in concentration, was depressed, refused to talk or eat and threatened suicide. He heard the voice of God commanding him to save the world, to become a Catholic and to take religious vows. He thought his father was responsible for his mother's insanity. He gradually improved and was returned to the United States about Oct. 1, 1918.

There were no physical symptoms.

By the middle of December, 1918, he showed no abnormalities except a slight retardation of thought and speech. Intellectually he appeared normal. The recommendation was then made that he be held until recovery was complete and discharged on certificate of disability as not in line of duty.

The nineteen cases of psychoneurosis consisted of thirteen of neurasthenia, five of hysteria and one of psychasthenia. All but one of the patients with neurasthenia were enlisted white men between the ages of 20 and 31, only four of whom had seen overseas service. The symptoms developed as a rule during the convalescent period following influenza, and every case was characterized by considerable fatigue, or feeling of "weakness." Besides the fatigue, there were found tremors, numbness, hyperesthesias, dyspnea and mild depression. Four cases were recommended for discharge, eight held at general hospitals for longer observation and one returned to duty.

In the following case symptoms of fatigue with mild depression, insomnia and tremulousness when excited developed during convalescence from influenza.

Case 10.—*History.*—Corporal O., aged 20, single, of mixed race, a student, had a neurotic mother. Little information concerning the personal history was available. He had reached the second year in college when he entered the army, enlisting Jan. 12, 1918. He had had measles, mumps, pertussis, and four attacks of pneumonia.

Present Illness.—This dates from Jan. 8, 1919, during convalescence from influenza, the latter lasting from December 15 to December 30. His chief symptom (subjective) was a complex characterized by physical and mental fatigue and attended by a host of other symptoms, mostly subjective and for which there seemed little basis. He was somewhat depressed and introspective. He appeared to be nervous and was easily excited, in the latter state being tremulous and weak. He easily tired from mental or physical effort. He was irregular in sleep, dreaming much. He was under observation for several months and was transferred to a general hospital for further observation.

The physical examination was negative. There were no organic symptoms.

This soldier contracted influenza and pneumonia in France with subsequent fatigue symptoms. After several months of treatment, he was restored to duty.

Case 11.—*History.*—Private S., aged 25, single, of mixed race, a farmer; his sister had "nervous breakdown." As a child he had measles, mumps, and whooping cough. He was of a rather low grade mentally, never advancing above fourth grade in school. He denied syphilis, alcohol and drugs. The total length of his service was one year and ten months, one year of which was spent overseas.

Present Illness.—While in France he had influenza and pneumonia followed by nervousness, weakness, dyspnea on exertion and rapid heart action. Examination revealed no organic heart lesion or other physical cause for symptoms. The patient appeared to have considerable insight, attributing his disability to influenza and pneumonia. After several months of treatment he was restored to duty.

As already stated, five cases were diagnosed as "hysteria." The patients were between 20 and 32 years of age; all but one were single, and all were white men. Three had served overseas. Among the symptoms noticed were weakness and numbness of the extremities, tremors and anesthetic areas, all without an organic basis. All of the patients were regarded as having been disabled in line of duty. Two were recommended for discharge, the others remained under observation and treatment in general hospitals.

The following patients suffered from weakness and numbness of the right hand and arm which ushered in an attack of influenza.

Case 12.—*History.*—Private J., aged 23, single, a farmer. English, had a negative family history so far as could be ascertained. Information was meager. He reached the fifth grade in school. He was not addicted to the use of alcohol or drugs. He served two months in the United States and four months in France.

Present Illness.—While in France, in October, 1918, he had an attack of influenza which was ushered in by weakness and numbness in the right hand and arm. This weakness and numbness continued a number of months, the soldier being still under special treatment in a general hospital in this country in February, 1919. He was quiet and well behaved, showing no intellectual defects or organic physical conditions to account for his symptoms. He said that electrical treatment which he received in France had benefited him greatly.

Several soldiers who had been in the army for some time and had apparently been able to give satisfactory service or who at least had not been brought to the notice of medical officers for inability to carry on their work, were found to belong to a group disqualified for service by reason of chronic and long existent neuropsychiatric conditions. This disqualification had apparently not been realized or perhaps not brought to notice until the men had contracted influenza and had been under close observation in a hospital ward. Among these patients were two cases of constitutional psychopathic state, one diagnosed "emotional instability," the other "inadequate personality."

Case 13.—The former a single white man, aged 21, a private, had served a total of two years in the army, eight months of which had been in France. Prior to army service, he had worked in many different positions, frequently changing for no definite reason. While in the army, he was absent without leave a number of times and was unable to adapt himself, being continually in conflict. Following the attack of influenza, his restlessness appeared to be aggravated and he was finally recommended for discharge from the army.

Case 14.—The patient with "inadequate personality" was a private, 28 years old, single, formerly a laborer, who had had ten months of service, six months overseas. During an attack of influenza in France, he had considerable delirium with periods of marked excitement. Following his return to the United States, he had difficulty in remembering and there seemed to be some "thought-blocking" which he appreciated, attributing his disability to influenza.

Two other cases were those of patients with mental deficiency of the moron grade.

Case 15.—A private, white, single, 30 years old, formerly a farmer, with a total of eight months' service, seven in France, during which he had influenza, showed mental dulness and emotional instability. By the Stanford-Binet test, his mental age was 7 years and 9 months. He was recommended for discharge from the army, not in line of duty.

Case 16.—A private, white, single, aged 26 years, formerly a teamster, had been in the army fifteen months, eight of which were spent overseas. Following an attack of influenza with pneumonia and pleurisy, he was very weak and depressed; later, as his physical conditon improved, he became rather boisterous and happy. A psychometric examination made his mental age 10 years and 4 months. Perhaps a modified diagnosis, such as a manic-depressive episode, should be considered in this case.

There were two cases of paralysis of the facial nerve, one of both sides, the other of the left side, which seemed to be quite definitely associated with attacks of influenza. Both soldiers made good recoveries and were returned to duty.

Two soldiers developed neuritis, one during an attack of influenza, the right brachial plexus being affected, the other, two weeks following the influenza, had neuritis of the right peroneal nerve. In connection with the brachial neuritis the chief symptom was weakness of the right arm accompanied by diminished reflexes and some pain or pressure over the nerve trunks and muscles. The disability in the other case was greater. The patient could not lift the right foot from the floor for about two months; weakness of the leg persisted still longer. There was general weakness in the right leg, accompanied by tingling and inability to raise the foot (foot drop).

There were two cases of cerebral embolus associated with influenza.

Case 17.—A recruit, who had been in the service only a short time, aged 21, single, a farmer, had no history or evidence of syphilis. He used alcohol moderately. Subsequent to a period of unconsciousness from an attack of influenza followed by pneumonia, he complained of weakness of the entire left side of the body and difficulty in using the left arm and leg. All deep reflexes were exaggerated, those on the right side markedly so, and there was left ankle clonus. The gait was typically hemiplegic. Sensation was unaffected.

Case 18.—The second case was that of a private, 26 years old, single, a farmer, who had been in the service three months. Twenty days after the onset of influenza and ten after pneumonia, he developed paralysis of the right arm and leg, with inability to talk. The paralysis was spastic, the deep reflexes being markedly increased; abdominal reflexes were absent on the right side; there was a suggestive clonus of the right ankle. The Babinski reflex was not present. Sensation was blunted on the entire right side.

The tremor in a case of hyperthyroidism was reported as being "increased by army life and the after-effects of influenza. Accompanying this tremor was a general nervousness elicited by any physical or mental stress, with rapid heart action."

COMMENT

Considering the large number of cases of influenza occurring in the army during the epidemic, it is somewhat surprising that more instances with a possible influenzal etiology were not found among the 70,000 neurosychiatric cases reported to the Surgeon-General. This would seem to indicate that mental and nervous complications of influenza, severe enough to require the attention of specialists in neuropsychiatry, were comparatively rare. It is generally conceded, however, that some degree of nervous or mental complications, such as slight neurasthenic or depressive symptoms, are rather common, but do not usually outlast the influenza process.

No attempt has been made to revise diagnoses after a critical consideration of the case histories. In fact, a majority of the records contained sufficient descriptive data to warrant a continuation of the diagnosis reported and all cases were examined, classified and treated under the supervision of competent neuropsychiatrists.

CONCLUSIONS

1. Among over 70,000 neuropsychiatric cases reported to the section of neurology and psychiatry in the office of the Surgeon-General, in only 73 could influenza be deemed a significant etiologic factor.
2. Thirty-two cases belonged to the infective-exhaustive-toxic group, nineteen were psychoneuroses, four manic-depressive psychoses, seven dementia praecox, four neuritis and seven miscellaneous.
3. The infective-exhaustive-toxic types presented delirious features and other symptoms indicative of a severe general reaction with a favorable outcome as a rule.
4. Several of the dementia praecox cases had apparently been normal mentally until the attack of influenza which seemed to be a definite etiologic factor.
5. The manic-depressive cases presented no unusual features.
6. Neurasthenia was the most frequent psychoneurosis, with the characteristic fatigability or feeling of weakness.
7. Influenza appeared to be a definite factor in bringing out or emphasizing difficult personal peculiarities in men who had previously been able to attend to their duties.
8. The neurologic conditions associated with influenza were paralysis of the facial nerve, neuritis and cerebral embolism.

SIGNIFICANCE OF FACIAL PAIN IN DETERMINING THE LOCATION OF INTRACRANIAL TUMOR*

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Pain and anesthesia of the face preceding the onset of deafness possess a significance that is not always appreciated. These symptoms are usually caused by a tumor involving the gasserian ganglion. Neoplasms of the gasserian ganglion invariably give rise to pain or cause objective disturbances of sensation; these symptoms appear early and precede the onset of deafness, or at least they are manifest before deafness becomes discernible.

The gasserian ganglion lies on the anterior surface of the petrous portion of the temporal bone, and is, therefore, situated within the middle cranial fossa; it is separated from the posterior fossa by the dura, which is firmly attached along the crest of the petrous portion of the temporal bone. Furthermore, the ganglion is enveloped above and below by the dura itself. For these reasons fibromatous tumors growing from the acoustic nerve in the cerebellopontile angle generally give rise to deafness as an early symptom and are not prone to extend far enough forward to encroach on the structure lying within the middle cranial fossa.

I have been unable to find records, drawn from reliable sources, of a single case of tumor arising from the cerebellopontile angle in which disturbances of hearing did not constitute an early sign. If this symptom does not occur at some time during the course of the disease, the diagnosis of tumor in the cerebellopontile angle is rendered extremely doubtful.

In an article on intracranial tumors published in 1916,[1] I referred to the importance of ascertaining the exact order in which symptoms arise in cases of intracranial tumor, and called attention to the fact that at times a particular type of tumor is encountered that grows from the dura at the base of the brain in the middle and posterior cranial fossa, frequently involving the bones of the skull; it does not infiltrate the brain substance, but does involve the cranial nerves. Tumors of this type are generally endotheliomas and are occasionally associated with cystic formations. They may arise from the region of the gasserian ganglion and extend back, appearing as a flat, slowly growing mass, subsequently involving the seventh and eighth nerves, giving rise to symptoms that resemble those of true fibromatous tumors, which spring from the eighth nerve in the cerebellopontile angle. Unlike these tumors, however, pain in the face is a conspicuous symptom.

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* Read at the Forty-Sixth Annual Meeting of the American Neurological Association, June, 1920.

[1] Cadwalader, W. B.: Intracranial Tumors, J. Nerv. & Ment. Dis. **44**:11 (July) 1916.

Owing to the tendency of these tumors to infiltrate the temporal bone, attempts at removal can hardly be attended with success.

REPORT OF A CASE

The case report presented is instructive because it tends to confirm the correctness of the deductions made from my former observations. Pain in the face followed by anesthesia, or anesthesia dolorosa, in the fifth nerve distribution was the most striking clinical symptom, and proved to be the most important localizing sign. Tinnitus and partial deafness developed later. Symptoms of intracranial pressure were absent. A diagnosis of intracranial tumor growing from the region of the gasserian ganglion was made. Operative treatment was considered, but in the light of experience was deemed inadvisable, because it would probably be impossible to remove a slowly growing, flat tumor mass, which is likely to be intimately connected with the meninges at the base of the brain in this region.

At necropsy the growth was found to occupy the suspected area: the tumor had embraced completely the root of the fifth nerve, and had extended downward and backward almost to the foramen magnum; it had also involved the temporal bone. The brain was not affected directly. Microscopic examination revealed the characteristic appearance of a chondroma. The cause of death was miliary tuberculosis.

History.—Mrs. A. K. was admitted to the University Hospital, Aug. 20, 1919, complaining of attacks of severe pain in the left side of the face. She stated that in February, 1919, she was suddenly seized with severe pain that involved the entire left side of the head and the middle and upper portions of the left side of the face. The pain did not extend to the chin, upper lip, tongue, or the inside of the mouth. The attacks of pain were infrequent during the day, but at night the paroxysms increased in frequency so that the patient had much difficulty in securing sleep. During the next few months the pain gradually became less severe and the attacks grew less frequent, until June, 1919, when it ceased entirely, but was replaced by a peculiar sensation, limited to the previously painful area, and described by the patient as a "running up and down sensation," or a feeling as if ants were crawling over the face. This was constant and particularly marked in the malar region. Occasionally these abnormal sensations became most intense and were accompanied by a feeling as if the mouth were drawn to the left; actual pain was also complained of. Later the patient described the pain as being "burning" and "sticking" in character, and involving, in addition, the left half of the tongue. Not long after the pain set in the patient complained for the first time of a roaring sound in the left ear. This increased in intensity and in June, 1919, it had become so constant as to be extremely annoying and distressing.

Examination.—Examination on admission disclosed the fact that the pupils were small but equal in size and contracted normally to light and in accommodation. The media were clear, and the disks oval and normal in color and outline. No fundus changes were apparent, and no extra-ocular palsies were present. None of the muscular movements of the left side of the face could be as well performed as on the right side. There was a decided weakness of the left facial muscle, but the paralysis was incomplete. Cutaneous sensation for touch, pain and heat was greatly diminished, but not completely lost in the entire distribution of all three branches of the left trifacial nerve. The conjunctiva and corneal reflexes were nearly absent on the left and normal on the right side. The motor branch of the left fifth nerve was also affected, for the left temporal and masseter muscles were distinctly weaker than the right. Hearing was reduced. The bone conduction was far better than the air conduction. The right ear was normal. A vibrating tuning-fork placed on the midline of the skull was heard better with the right ear. The upper and lower limbs and the tendon reflexes were normal.

A subsequent examination, made less than a month before the patient's death, showed that general weakness had increased greatly; the patient was confined to her bed, lying generally on the left side. She was stuporous, but could be roused. A constant buzzing in her left ear and a sensation of vibration in the head were complained of. Headache was not severe. The left external rectus was completely paralyzed. The left eyeball was more prominent than the right. The anesthesia of the left side of the face had not changed. Four days before death the patient complained greatly of tinnitus, and was almost, but not completely, deaf in the left ear. The tendon reflexes were increased. She died Dec. 5, 1919.

Postmortem Examination.—On the upper aspect of the petrous portion of the temporal bone there was a swelling firm in consistency and grayish in color. It extended from the apex of the petrous bone for a distance of 2.5 cm. toward its base, and from the sharp edge of the bone 2 cm. into the temporal fossa. Nowhere was the tumor elevated more than 2 cm. above the bone. Immediately under the surface of the temporal bone, near its apex, there was a tumor of similar gross appearance, but measuring 12 mm. in length and 10 mm. in its greatest width; this tumor extended about 4 mm. above the surface. A similar swelling the size of a split-pea was found at the extreme lower border of the petrous bone, half way between the extremity and the base. The dura mater was slightly adherent to the lower border of the temporal bone, the adherent area being about 10 mm. in diameter. On outward inspection a slight flattening of that part of the left temporal bone that overlies the left petrous bone was visible, but otherwise no external changes in the brain were evident. Springing apparently from the left petrous bone was a tumor elevated about 3 mm. above the bone surface; it appeared to be well circumscribed, and extended from the apex to a point midway between the apex and the base, and from the anterior edge of the petrous bone to the foramen magnum below. The tumor measured in detail: The section of the growth on the anterior portion of the petrous bone was 25 mm. in length and 20 mm. in width, and was roughly quadrangular; the portion of the tumor on the lower surface of the petrous bone was also roughly quadrangular in shape, and measured 40 mm. in length and 32 mm. in width. Near the apex of the petrous bone the tumor was somewhat more prominent and assumed the shape and size of a bean. The fifth nerve made its exit from the tumor mass. The growth was of a pale, grayish-white hue and was firm in consistency. It extended almost to, but did not quite touch, the pituitary body.

The tumor invading the surrounding bone was chiseled out, and it was found that the neoplasm extended into the antrum, where a firm, whitish, cartilaginous growth, about the size of a hazelnut, was found.

ENDOCRINAL DEFECTS AND MENTAL STATES

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A recent article by Kempf [1] on "The Tonus of Autonomic Segments as Causes of Abnormal Behavior" says "The ascent of man from a relatively simple organism (the embryonic cell) that is, a complete autonomic apparatus, which specializes into systems and gradually develops a striped muscle or projicient apparatus, naturally divides the matured organism into an autonomic apparatus and its projicient apparatus. The autonomic apparatus is constituted of the organs and ganglionic nervous systems which regulate the assimilation, conservation, transformation, distribution and use of the energic products which are necessary to sustain and promote life and the elimination of the waste products. . . . The projicient is constituted of the skeleton and striped muscle systems, the exteroceptors, the intramuscular proprioceptors and their cerebrospinal nervous systems. . . . At birth we have a well-developed harmoniously reciprocating autonomic apparatus and a poorly coordinated projicient apparatus, but the autonomic apparatus begins immediately to coordinate and exercise control over the projicient apparatus in order to master the environment. This most important factor begins to exert pressure upon the infant at birth and continues throughout its life." Not every instance of such control by the autonomic apparatus has been given physiologic demonstration, but the work of Sherrington is quoted as proving the principle. Kemp says. "It is sufficient to know that in a quick continuous intimate manner our autonomic effective tensions influence the general tone of our striped muscle and this tonus by stimulating the proprioceptive system of the striped muscle apparatus, determines the nature of the kinesthetic stream, hence, largely, the content of consciousness. . . . Disturbances in the autonomic apparatus if not readjusted must seriously jeopardize the individual's biological career and self-control in his struggle for life and social esteem."

ENDOCRINAL DEFECTS OF APPEARANCE

This quotation serves as an introduction to a few considerations regarding endocrinal influences on mental conditions as disturbances in the autonomic apparatus are a feature of endocrine control. To put it another way, the person who has a perfect endocrinal balance would show no disturbance of the autonomic apparatus. The endo-

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[1] Kempf: Jour. Nerv. & Ment. Dis. **51**:1 (Jan.) 1920.

crine chemistry of the individual conditions the autonomic apparatus and that produces the content of consciousness including emotion, and this in terms of perpetuation and external projection is personal character. But there is another point to be thought of, and this phase is the one that I shall speak of chiefly. The endocrine factor not only affects the mental condition or personality through an immediate direct chemical fluctuation by the way of the so-called autonomic segments — such an immediate endocrinal influence as is described in Kempf's analysis — an influence which can be called chemically combustible if one may use that metaphor in order to emphasize the urgency of the situation; but, in addition, the endocrinal factor figures in a static and less direct manner. It does this through the mediation of numerous endocrinological defects, some of which are: gigantism, dwarfism, ungainly length of arms, ungainly proportion of trunk and leg, acromegalic hands or feet, acromegalic features, a cretinoid face, a gerodermic skin, a Froehlich's dystrophy adiposity, a status lymphaticus infantilism, a difiguring facial hypertrichosis in a woman, an underslung jaw, protruding upper teeth, a disfiguring acne, a falsetto voice in a man, a swollen and distorted neck and thyroid struma and even bulging eyes. Such a list may seem bizarre, but I believe it is worth while to make it and to think of such personal features as a neglected source of endogenous factors in abnormal mental states. They become so through being the source of perplexities, doubt, discouragement and feelings of inferiority, and weigh heavily in many transactions of the psyche. I would hasten to add that I do not imply that they are all of equal valence. Some would cause no annoyance in a man, but would be potent in a woman. For example, unnatural height would not disturb the man as much as the woman, and the reverse would be even more definite in the case of unnatural shortness of stature. Also, they would not produce the same mental discouragement at all periods of life. For example, for a young woman a disfiguring facial hypertrichosis can scarcely be overemphasized as a source of gnawing and harmful worry. It probably grows less so as she advances into middle life, but I doubt if it disappears until she is actually in the period of senility. The effect of such a feature is augmented through being the sort of thing which is not talked about freely — it is taboo. The resultant repression and its influence need only be mentioned. Others of the so-called endocrinal defects which I have mentioned bring in these factors of silence, repression and increased affect in a similar way.

ENDOCRINAL DEFECTS OF FUNCTION

Defects of function are also important. They are at the same time dependent on immediate endocrinal influences working through the autonomic segments and on such defects as have been named made operative by introspection and worry. It is difficult to give examples of such defects of function because most of the examples are complex and clouded by other factors; but an idea of the meaning may be gained by taking as an example the underdeveloped boy. What of the poor record in pursuits in which the boy should excel? Does failure in sports in that stage of his development have no influence on his character? Again, what of the hypo-adrenal asthenic, or the thin-muscled person with status lymphaticus? Does the hypotonia of these types of individuals handicap them in athletic prowess? Does the feeling of inferiority which results from this handicap in an average American community have any bearing on that phase of mental growth which is dependent on deeds accomplished? Inherent physical incapacities cannot always be analyzed, but when they can be, a defect such as hypotonia, which is culled out in the process, may be regarded as an example of what is meant by an endocrinal defect of function. Another example is enuresis in childhood. I must express the belief that there is apt to be a hypopituitary factor in every case of enuresis (hypopituitarism means lack of tone of smooth muscle, and this occasions enuresis). This particular type of endocrinal defect of function is a probable source of grave psychic injury to the child through the reproach, disgrace and openly imputed inferiority which it occasions. I believe the reproach and imputations are the starting point for the majority of the psychogenetic factors found in these persons, and not the psychogenic factors the cause of the average enuresis.

Considered endocrinologically, in the individual it is the repeated and reiterated functioning of things along the lines of the theory of the autonomic segments plus the more static effects that I have endeavored to point out which produce personality. That there are various summated types of personality which are definitely the result of endocrinal predominances can hardly be questioned — such as the thyroid personality, the pituitary personality, and so forth. However, the refinement and description of these personalities will not be attempted in this paper.

ABNORMAL MENTAL STATES CAUSED BY ENDOCRINAL DEFECTS OF APPEARANCE OR FUNCTION

What types of abnormal mental states, if any, succeed the worry and discouragement which is caused by endocrinal defects of appearance or function? I believe they are often found in dementia praecox and psychoneurosis. The figures I have which correlate these two conditions with endocrinal defects are not yet matched by any regarding manic-depressive types. Therefore, I am omitting the latter types. I do not forget that involution melancholia is probably the most clearly established endocrinal psychosis, but because it does not show the mechanism of conscious worry over a clearly comprehended defect with the injury induced, it will not be included in this discussion. Since it seems preposterous at first glance to mention dementia praecox here when it is a matter of a mechanism involving first, defects, then worry, and implying sufficient insight to make the worry harmful in degree I hasten to add that it is in the stage before the dementia praecox exists that such a mechanism may be at work. In other words, it relates rather to the "shut-in" personality and stands as a conception to explain at least in part what lies back of such a personality. Hoch [2] says concerning dementia praecox, "We see, therefore, that the traits upon which we would lay most stress in the 'shut-in' personality, (are) the lack of contact with the environment, the satisfaction with fancies instead of objective interests, the lack of constructive aims and aggressiveness." There is a suggestive parallelism between these traits and those which would naturally arise if during adolescence the boy lacked normal physical aptness. One type of boy who often lacks such aptness is the flagrantly disendocrinal individual—the status lymphaticus case. Is it not possible that his withdrawal into himself and development of a "shut-in" makeup is a reaction to his endocrinal situation? Amsden [3] has written concerning the traits of the "shut-in" personality in dementia praecox: "The study of many cases convinces one that these traits are developed in relation to a vaguely felt deficiency." An obvious endocrinal abnormality is not apparent in every case of dementia praecox; but neither is the demonstrable "shut-in" personality. I should like to know — and only observation of a long series with the point in mind will tell — whether those cases which lack the "shut-in" personality or the shallowness of interest which, as Kirby [4] has shown, replaces it in some instances, are also the cases in which constitutional endocrinal disturbances, particularly status lymphaticus, hypoadrenalism and hypopituitarism, are not found. Figures regarding status lymphaticus are worth quoting. Emerson [5] found that status lymphaticus occurred in more than 29 per cent. of cases of dementia praecox, and I have found it in 24 per cent. of cases of war neuroses in a series of over 100.[6] As regards the frequent occurrence of this among the war neuroses cases, it does not seem surprising that the person with status lymphaticus whose frame was not stalwart, whose muscles were not developed, whose muscles because of a constitutional hypotonia lacked even the potential of normal development, did not stand up under the stress and exhaustion of army life. I do not think one looks for cases of status lymphaticus in college football teams, and men with such cases in the army played "football," to speak metaphorically, not more successfully in France. I would emphasize again the person's awareness of the defect as a factor.

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[2] Hoch: Constitutional Factors in the Dementia Praecox Group, Rev. Neurol. & Psychiat. **8**:463 (Aug.) 1910.

[3] Amsden: The Study of the Personality in Psychiatric Cases, Am. Medico-Psychological Assn., Seventy-Fourth Annual Meeting, June, 1917.

[4] Kirby: Not published.

[5] Emerson: A Note on the Incidence of Status Lymphaticus in Dementia Praecox, Arch. Int. Med. **14**:881 (Dec.) 1914.

[6] Davis: Status Lymphaticus: Its Occurrence and Significance in the War Neuroses, Arch. Neurol. & Psych. **2**:414 (Oct.) 1919.

I have recently seen an arrested case of acromegaly in a young man of 28 who now has symptoms of a severe psychoneurosis. This developed only after the acromegaly had reached a disfiguring stage. It has resisted treatment, and this resistance seems to be based on the threatened permanence of the cause of his distress — the acromegalic face. Having had no anamnesis from any one except the patient himself, I can neither bring complete proof regarding his initial makeup nor exclude the possibility that he offered a field for psychoneurotic developments. However, such evidence as we have is quite against that possibility, and his developmental history shows a considerable degree of initial normality. Now he is unable to meet either strangers or old friends; he has several times broken down and cried in public places because there his thick lips and unshapen jaw he feels certain are the cause of unfriendly comment. He shows deep emotion when telling any one of his trouble. He can initiate no plans because his mind is lost in thinking of his affliction; he has no longer the ambition or the confidence natural to him. This case calls our attention, I think, to the defect type of endocrinal influence.

I would like, then, to present the consciously comprehended physical defect as a common factor in the development of both psychoneurosis and dementia praecox, and add that in each up to a certain point, there is a similar mechanism, involving insight, worry, discouragement and alteration of character. True, such a mechanism is far from universal in these conditions, as is the initial defect to initiate it. That sequence in which the worry, discouragement and its bad effects follow in order needs as its preventive education in the fullest sense of that word, so that a philosophizing self-knowledge may be reached. That such a factor as the endocrinal defect should be common to two such conditions as psychoneuroses and dementia praecox rests on the fact that in dementia praecox it is exceedingly early and, one might say, prodromal. I have meant to make that point prominent. In each, other factors enter — in the praecox to complete the estranging and withdrawal and in the psychoneuroses to produce what may be called a combative attitude. In fact, one might distinguish them in terms of fleeing and fighting. While up to a certain point the praecox and psychoneurotic patients face similar difficulties and receive similar ill effects, the former flees the situation and retreats into a psychosis, while the latter stands and fights, though ever so wildly.

SUMMARY

An attempt at a complete description of endocrinal influences in mental states would be far outside the limits of a single paper. In addition, I doubt whether all the facts bearing on this relationship are as yet known. For brevity's sake, therefore, while I have mentioned the type of endocrinal influence which is immediate and chemical, I have emphasized chiefly static factors based on endocrinal defects of appearance or function, whose bad effects are dependent on insight and discouragement, whose innocuousness is only gained through an educative process forestalling this discouragement. It is suggested that this second type of endocrinal influence is a not unusual early factor in both dementia praecox and psychoneurosis even though these two conditions can be sharply differentiated. In the case of the patient with praecox this reaction to an unsatisfactory and consciously recognized (endocrinal) situation would explain in some instances the "shut-in" personality, and appear to lie back of that makeup. In the praecox patient the subsequent developments amount to a fleeing from this situation, while among the psychoneuroses patients, broadly speaking, there is a wild (psychologic) combativeness and pragmatically less submission.

AN ANALYSIS OF CASES ADMITTED TO THE NEUROPSYCHIATRIC SERVICES OF U. S. ARMY GENERAL HOSPITAL NO. 1 (COLUMBIA WAR HOSPITAL, NEW YORK)*

SYLVESTER R. LEAHY, M.D.
BROOKLYN

On Nov. 22, 1918, Ward 55 of the U. S. Army General Hospital No. 1, was opened for the reception of neuropsychiatric patients arriving at the Port of Embarkation, Hoboken, N. J., from overseas and also for such cases as developed in the hospitals under the jurisdiction of this port. This had formerly been the Messiah Home and had been maintained for the care of children. It was leased from the directors of the Home by the Knights of Columbus and turned over to the War Department at a charge of $1.00 per year. The general construction was so good that with but a few alterations it was readily adapted for the class of patients with which we had to deal.

The building contained five wards, two of which were devoted to the frank psychoses, one for disturbed patients and the other for quiet, depressed ones. The remaining wards were used for the care of mild mental states, psychoneurotics, epileptics, constitutional psychopaths, etc. The hospital had a total bed capacity of 220. Of this number the ward for disturbed patients contained thirty beds, the ward for quiet patients forty beds, and the remaining three wards contained fifty beds each.

The staff consisted of an executive medical officer, chief of service, five ward surgeons, a mess officer, a registrar and a dental officer. The dentist had a complete outfit and inspected the teeth of all patients admitted. As this part of the general hospital functioned as an evacuation unit, only urgent dental conditions could be treated, but charts were made of all pathologic findings and recommendations for treatment were written thereon. This chart was then forwarded, with the history of the patient, to his final destination. In view of the fact that the unit was an integral part of General Hospital No. 1, we were able to arrange for consultation with the members of this staff and their services were always promptly and cheerfully available. As a result of such an arrangement, many patients actually ill with conditions other than mental could be immediately transferred for treatment.

The hospital was equipped with a complete hydrotherapeutic outfit, consisting of continuous baths, showers, needle spray and douche, etc.

An occupation class under the direction of a trained worker and three assistants completed the therapeutic system.

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* Read before the New York Neurological Society, April 6, 1920.

On admission all patients were immediately inspected for the internationally famous "cootie," venereal diseases and throat infections. Throat cultures were taken on all admissions. Following this procedure, a hot shower was given to all but "cootie patients," who were given a special tub bath. The clothing of all patients was sterilized by steam.

CLASSIFICATION OF PATIENTS

As soon as possible after admission a complete physical and mental examination was made. The cases were classified and reported to the office of the Surgeon at Hoboken, N. J., in order that transportation might be arranged. If the diagnosis on the field card accompanying the patient was not concurred in, the patient was presented at staff meeting and the consensus of opinion determined the diagnosis. In all doubtful cases, blood and spinal fluid examinations were made. In addition, ophthalmic, aural, surgical and medical examinations were made where there were special indications. Where a diagnosis of mental deficiency was in doubt, an intelligence test by means of the Stanford Revision was made and in many cases the diagnosis was changed. This cast no reflection on the work of the psychiatrists overseas, as many of these patients presented a far different aspect after reaching this country. The psychoses patients, too, often presented a far different appearance from that previously noted in their records, and although formerly indifferent and depressed, now presented a cheerful, interested aspect. The total number of patients admitted was 2,750, of which, 2,126 were overseas and 624 were local cases.

The patients admitted to the hospital were classified as follows:

TABLE 1.—Classification of Patients with Organic Nervous Diseases

Amyotrophic lateral sclerosis	1
Sydenham's chorea	1
Acute encephalitis	1
Lateral sclerosis	2
Tabes dorsalis	4
Multiple neuritis (following typhoid fever 1, diphtheria 2, intravenous administration of arsphenamin 1, alcohol 4)	8
Cerebral syphilis	33
Peripheral nerve injury	9
Gunshot wound of the head	2
Head injury without demonstrable fracture of the skull	7
General paresis	61
Fractured skull	7
Brain tumor	3
Progressive muscular atrophy	1
Myotonia congenita	1
Transverse myelitis following intrathecal administration of arsphenamin	1
Nervous disease undiagnosed	2
Total	144

It will be seen from the above that 100 of the 144 organic nervous cases were syphilitic diseases of the central nervous system.

The cases classified as "Observation for Epilepsy" presented no evidence in the accompanying history that a conclusion had ever been observed by a medical officer and as none occurred at this hospital, it was deemed fair to the patient to leave the diagnosis open.

TABLE 2.—Cases of Epilepsy and Observation for Epilepsy

Observation for epilepsy	25
Grand mal	111
Petit mal	9
Equivalents	1
Jacksonian	1
Traumatic	2
Psychosis	2
Total	151

The patients listed under the heading "Recovered" had usually had either a mild depression or excitement of the manic depressive type or else had had an acute alcoholic hallucinosis from which they had completely recovered. A number of psychoneurotics appeared to have recovered in that they were free from symptoms during their residence and so were placed in this group because it was felt that further hospital residence was unwise and might produce a recurrence of their symptoms.

Of the recovered cases the subclassifications were as follows:

TABLE 3.—Classification of Recovered Cases

Psychoneurosis	17	
Alcoholic hallucinosis acute	7	
Manic depressive psychosis	9	
Psychosis undiagnosed	1	
Alcoholism acute	1	
Pathologic intoxication	1	
Delirium tremens	1	
Psychosis following influenza (infective exhaustive)	3	
Undifferentiated depression	2	
Gunshot wound of the spinal cord	1	
Recovered	—	43

TABLE 4.—Mental Deficiency and Manic Depressive Psychoses.

Mental deficiency with psychosis		4
Manic-depressive psychoses:		
Manic type	95	
Depressed type	213	
Mixed type	44	
Circular type	1	
Manic-depressive psychosis	——	353

In the manic-depressive psychoses group, in so far as we were able to obtain reliable information, thirty-five had had a previous attack. It must be remembered, however, that the number of patients who had

had previous attacks was undoubtedly greater, but as many of the patients were entirely inaccessible, information in regard to this could not be obtained. The depressions predominated.

TABLE 5.—Cases of Dementia Praecox

Hebephrenic	256
Paranoid	163
Simple	111
Catatonic	20
Total	550

Many of the patients presented a typical schizophrenic history, but were in an apparently normal condition and well adjusted. Some of them gave quite adequate explanations for their upset, such as nostalgia and worry over misfortune at home. Others stated that they felt they had been unfairly treated in the army. The eventual outcome appeared to be problematical. We felt that the original diagnosis should be left unchanged.

TABLE 6.—Cases of Paranoid Condition, Alcoholic and Traumatic Psychoses, Constitutional Psychopathic State and Psychoneuroses

Paranoid condition		4
Psychoses with somatic disease:		
Following influenza	20	
Following mumps	2	
Following pneumonia	1	
	—	23
Traumatic psychosis (head injury)		5
Alcoholic psychoses:		
Acute hallucinosis	37	
Deterioration	4	
Pathologic intoxication	1	
	—	42
Constitutional psychopathic state:		
Inadequate personality	110	
Emotional instability	8	
Paranoid personality	5	
Delinquent tendencies	1	
Homosexuality	3	
Criminal tendencies	3	
	—	130
Psychoneuroses:		
Hysteria	295	
Neurasthenia	282	
Psychasthenia (compulsion neurosis)	25	
Anxiety state	22	
Hyperthyroidism	8	
Enuresis	8	
Disordered action of the heart	5	
Traumatic neurosis	3	
Stammering	6	
Syphilophobia	2	
Hypothyroidism	2	
Somnambulism	1	
Dyspituitarism	1	
Facial tic	1	
	—	661

Of the neurasthenic group 26 per cent. of the patients gave a history of having had symptoms of this condition in civilian life and of the hysteria group 19 per cent. gave a history of similar trouble prior to army service.

TABLE 7.—Cases of Inebriety

Alcoholism	111
Morphin addiction	7
Heroin addiction	6
Heroin and morphin addiction	1
Total	125

The small number of drug addictions is noticeable.

TABLE 8.—Cases of Mental Deficiency, without Mental Disease and Undiagnosed

Mental deficiency:		
Morons	225	
Imbeciles	27	
		252
No mental disease found:		
Rheumatic fever subacute	1	
Flatfoot	1	
Duodenal ulcer	1	
Pulmonary tuberculosis	3	
Syphilitic cirrhosis of the liver	1	
Gastritis, chronic catarrhal	1	
Syphilis, secondary	1	
Acute gonorrhea	1	
Deviated nasal septum	1	
Diphtheria carrier	2	
Malaria, tertian	1	
Polyarthritis, rheumatic	1	
Gunshot wound of the right arm	1	
Valvular heart disease	1	
Diagnosed as epilepsy but not concurred in	1	
No physical or mental disease found	13	
		31
Psychoses undiagnosed		148

The cases with psychoses undiagnosed were left ungrouped because of the lack of data sufficient to make a differentiation possible. Many of these patients were fearful and refused to answer questions. They were not catatonic nor did they attitudinize. Hallucinatory reactions were not observed. Other patients appeared quite confused and presented a dreamlike perplexed state. At times they appeared quite depressed. They refused to cooperate on examination. Many of the patients were difficult to differentiate adequately, and it could not be definitely decided as to whether they presented a praecox or manic depression reaction. In many cases there was an alcoholic history and coloring which was difficult to properly evaluate. In a few of the cases there were pupillary signs, but the residence was too short to permit of blood and spinal fluid examinations, or else they were too disturbed for such procedures.

This part of the U. S. Army General Hospital No. 1, closed officially on Sept. 10, 1919, but no patients were received after Sept. 1, 1919, so that it was open for the reception of patients for a period of nine months and twenty-two days.

COMMENT

1. None of the cases appeared different from those encountered in civilian life, except that most of them had a military coloring.

2. Of the total number of 2,750 patients, 24 per cent. were psychoneurotics, 20 per cent. of the dementia praecox type, 12 per cent. were of the manic-depressive group, 10 per cent. mental defectives, 5 per cent. had organic nervous diseases, principally of the syphilitic type, 4 per cent. were definitely epileptic and 4 per cent. were constitutional psychopaths.

3. There were only fourteen cases of drug addiction, or about 0.5 per cent. of the total admissions.

4. Many of the cases apparently of the praecox type appeared to be recovered with excellent insight.

5. Of the neurasthenic group, 26 per cent. of the patients gave a history of having had symptoms in civilian life, and of the hysteria group 19 per cent. gave a history of similar trouble prior to army service.

A comparison of the group percentages found at this hospital with the group percentages of the total male admissions for the New York State Hospital service during the year 1919, is interesting. During this year the total first admissions were 6,791. Of this number, 3,527 were men. The group percentages for the male admissions are as follows:

TABLE 9.—Classification of Men Admitted to the New York State Hospital During 1919

	Per Cent.	Number of Cases
Traumatic psychoses	0.5	18
Senile psychoses	9.0	324
Cerebral arteriosclerosis	6.0	236
General paresis	20.0	710
Cerebral Syphilis	0.4	15
Organic brain diseases (Huntington's chorea, brain tumor, etc.)	0.5	19
Alcoholic psychoses	5.0	204
Drug psychoses	0.16	6
Psychoses with somatic disease	2.0	76
Manic-depressive psychoses	9.0	352
Involution melancholia	1.5	56
Dementia praecox	27.0	1,001
Paranoia	1.5	59
Epileptic psychoses	2.7	96
Psychoneuroses	1.0	35
Constitutional psychopathic state	1.6	66
Psychoses with mental deficiency	2.5	88
Psychoses undiagnosed	3.9	138
Not insane	0.7	28

While a strict comparison is not possible, it is interesting to know that there is a close ratio between the percentage of cases of dementia praecox, namely, 20 per cent. in the army and 27 per cent. in civilian life, and between the percentage of cases of manic-depressive diseases, 12 per cent. in the army and 9 per cent. in civilian life. Dementia praecox in both instances forms the largest group of the psychoses. Comparisons between the other groups is impossible because the civilian state hospitals deal primarily with psychoses occurring at all ages and with unselected population. In 1918, of the total remaining population in the New York State hospitals, 59 per cent. were of the dementia praecox group. It will readily be seen, therefore, that our great problem, from the standpoint of psychoses, both civilian and military, is that of the dementia praecox group. If it were possible to trace these patients, the eventual outcome would be interesting and might throw some light on this perplexing problem.

Grateful acknowledegment is hereby made to the men who served on this service, for their work and help in classifying these cases.

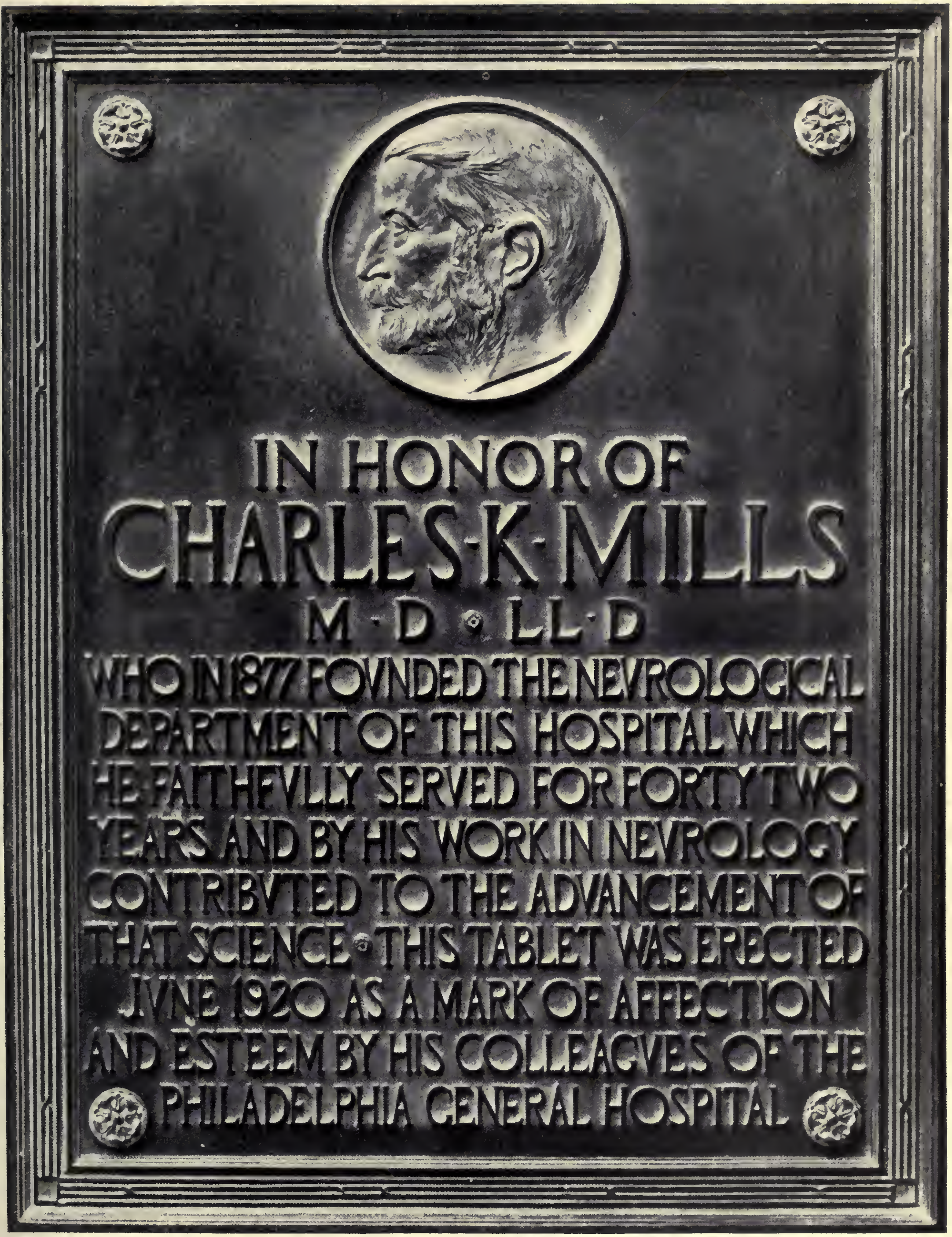
ADDRESS AT THE DEDICATION OF A TABLET IN HONOR OF DR. CHARLES K. MILLS

AT THE PHILADELPHIA GENERAL HOSPITAL
JUNE 17, 1920

JAMES HENDRIE LLOYD, M.D.
PHILADELPHIA

We have met today to do honor to a friend and colleague whose name will always be associated with the beneficent work which for so many years has been the distinction of the Philadelphia General Hospital. This tablet is the symbol of that association—but it is only a symbol, for the work itself, in its far-reaching influence and effects, is more enduring and more worthy of commemoration than can even be expressed in a tablet of bronze. It is said that Charcot took the chronic and neglected cases of the Salpêtrière, and on his study of those cases helped to lay the foundations of the modern science of neurology. With an insight no less wise and discerning than that of the great French neurologist, did Dr. Charles K. Mills see and grasp the opportunity to have established in this old city hospital a department of nervous diseases, which has won its place as one of the foremost neurological clinics of the world. This suggests our theme—which will be a tribute alike to the hospital and the man.

From the beginning the Philadelphia Hospital had suffered some lack of prestige from being looked on by the public as merely an almshouse. The title, as applied to the hospital, was a misnomer, for Blockley has always been much more than an almshouse; it has been a large general municipal hospital, including in that title also a large hospital for the insane. Not, however, until the formal establishment of free clinical teaching in 1860, and the erection of the clinical amphitheater in 1861, can the present era of the hospital be said to have begun. Before that date, however, as you can read in the late Professor Agnew's historical paper, a long record carries the Philadelphia Almshouse and Hospital far back into colonial times, for this institution, as Dr. Agnew showed, is the oldest hospital in the United States. During that long period it had twice changed its site, until, about 1830, it was located where it now stands; and it had an interesting and checkered career as a seat of clinical instruction. It numbered on its staff some of the most illustrious names in the medical profession in America; it admitted to its wards, on the payment of a good round fee, medical students who came from many parts of the United States; it harbored a great population of the insane,



IN HONOR OF
CHARLES·K·MILLS
M·D·LL·D
WHO IN 1877 FOVNDED THE NEVROLOGICAL
DEPARTMENT OF THIS HOSPITAL WHICH
HE FAITHFVLLY SERVED FOR FORTY TWO
YEARS AND BY HIS WORK IN NEVROLOGY
CONTRIBVTED TO THE ADVANCEMENT OF
THAT SCIENCE · THIS TABLET WAS ERECTED
JVNE 1920 AS A MARK OF AFFECTION
AND ESTEEM BY HIS COLLEAGVES OF THE
PHILADELPHIA GENERAL HOSPITAL

Bronze tablet dedicated to Dr. Charles K. Mills at the Philadelphia General Hospital. Unveiled June 17 with a dedicatory address by Dr. James Hendrie Lloyd.

some of whom, I regret to say, it kept in cells and straight jackets; it gave birth in its obstetric wards to more than 2,000 children in seven years; it had one of the largest specific services in the land; its medical and surgical clinics were conducted, from pre-Revolutionary days to the days of and after the Civil war, by such men as Bond, Dorsey, Rush, Physick, Gibson, Chapman, Stillé, Penrose, Agnew, Da Costa, Gross, Pancoast, Pepper, Tyson, Wood, Musser, White, Osler and others too numerous to mention; but in all that time it contributed little if anything to neurological science.

It was, indeed, a great hospital—a world within itself; with a population sometimes exceeding 4,000 souls. It was, and is today, one of Philadelphia's unique institutions, with a long history of service to humanity and to science, an honor to the city, the seat of some of the best medical traditions and associations in our midst.

But much of the clinical material gathered within the walls of Blockley was, from the very nature of things, of a most unpromising kind. In its vast population of the indigent, the infirm, the hopeless, there was a great preponderance of chronic and incurable cases. Gathered together from the far reaches of a great city, these chronic patients were the wards of charity rather than the objects of scientific interest. The public conscience was satisfied when it provided for them a last refuge on earth; the public debt was discharged when it gave to the paralytic a cot to sleep on and three meals a day. Whether it was with the inspiration of genius or simply with good, hard common sense, Dr. Mills saw the necessity and the opportunity for the establishment in the Philadelphia Hospital of a special department for nervous diseases. The idea was new, original, so far as this hospital was concerned—and, in fact, so far as any large hospital in Philadelphia or the country at large was concerned, for neurology as a specialty had hardly advanced that far. Weir Mitchell, indeed, had made a fruitful start in the comparatively small Orthopedic Hospital, where he was developing a nervous infirmary which was destined to be associated with his name and work, but the old idea was that a big hospital was naturally divided into a medical and surgical (and possibly an obstetrical) department, for nervous diseases were not *sui generis*—they belonged to medicine, and medicine had never made much out of them in Blockley. It is doubtful whether a good neurological paper had ever been written from this hospital before that time.

It was in 1877 that Dr. Mills won the consent of the old Board of Guardians (by whom the hospital was then administered) for the establishment of a neurological department. He had been appointed neurologist to the hospital in September of that year, the first position of the kind in Philadelphia and probably in the country. He has the distinction, therefore, of having first won official recognition for our specialty—an instance of his own primacy and the primacy of this hospital in the advancement of neurology.

The first patients—men and women—placed under his care in the wards for nervous diseases were less than twenty in number, and they were brought from various regions of the hospital where they had been receiving little if any attention. But the growth of the service was rapid, until in 1898 the number of patients amounted to more than 500, and in more recent years this number has been maintained or even exceeded. From having had at first only one neurologist, the staff has increased until it now numbers eight members, with eight assistants. The psychopathic department, which is practically an offshoot from the neurological, has also its separate staff.

The neurological department of the Philadelphia Hospital has thus developed into a great special service—and it is not too much to say that it has been in a sense the headquarters of Philadelphia neurology. If there is such a thing as a Philadelphia school of neurology, it is the Blockley school, for without exception all the men in this city now living who have attained distinction in neurology, have served on its staff. It has been the foster mother of the Philadelphia Neurological Society, for without its nourishing aid the transactions of that society would have been small indeed. It has given their earlier training to men who have been called to professional chairs in our various colleges, and supplied many of the themes and illustrations of those whose writings have done much indeed to keep Philadelphia in evidence as a neurological center. As of old, it has been a teaching hospital, and medical students have been initiated here into the mysteries of what is generally conceded to be the most difficult of the specialties. The facilities offered by its wards for the study of organic diseases of the nervous system are unsurpassed in this country, and scarcely surpassed abroad. In 1888, when Dr. Ferrier and Mr. Victor Horsley, both of whom were distinguished for their original work, came to America and visited Blockley, they said it reminded them more of the Salpêtrière than any hospital they had seen in this country.

During the recent great war a school of neurology was established by the United States government in Philadelphia for the benefit of medical officers entering the army and navy, and a large part of the teaching was done in the wards for nervous diseases of the Philadelphia Hospital by members of the staff, or in their clinics in other hospitals.

Graduate instruction has also been given for some years, and the graduate school, which was originally proposed by Dr. Mills, has only recently been practically merged in that of the University.

In its long career the Philadelphia Hospital has sometimes been a storm center of great magnitude. The waves of criticism have rolled over it, but it has usually emerged with renewed life. In these crises no one more than Dr. Mills has stood for improvement, and against any policy of concealment or stand-pattism, but he has also never been slow to champion the hospital. In sympathy with his wise leadership the members of the neurological staff are loyal to Blockley, and have always cultivated an *esprit-de-corps*. All they ask for the hospital is a fair deal. They believe that the work done here, both in neurology and psychiatry, equals if it does not surpass, both in quantity and quality, the output of any other hospital in this city. Our buildings may be old, but our science at least is up to date. Under the recent administration of Dr. Wilmer Krusen, the interests of the hospital have been greatly advanced and we look forward with confidence to a period of improvement under the administration of Mayor Moore and Dr. Furbush, which will advance the hospital to a still higher plane, and will also continue for the neurological department its era of prosperity and distinction which was inaugurated more than forty years ago by Dr. Charles K. Mills.

This is hardly the time or the occasion for an elaborate review of Dr. Mills' work in his chosen specialty. His name is so closely identified with that specialty in this country that an account of the one could not be given without a history of the other. But a fair and candid eulogy need not swell to the proportions of a history, for we are concerned today to link his name and fame with the record of old Blockley rather than to trace his work too far afield. We know how long and faithfully he has served the University of Pennsylvania in the chair of neurology; how he has written a textbook of nervous diseases which bears on every page the impress of his wide knowledge and experience; how he has contributed to medical literature more than two hundred special papers on all phases of neuropathology; how he has enriched the proceedings of our various medical societies and been honored with membership in learned societies abroad. In medical jurisprudence he has an established reputation in our courts; in consultation, a high repute whether at the bedside or the council board. All these are the evidence and the fruitage of a long career devoted to the advancement of neurological science.

But in our gathering here today, in our dedication of this tablet, there is something more in evidence than a mere impulse to do honor to the physician and the scientist. We pay our tribute of affection to the man. Our colleague, our friend, our chief, we salute and applaud him.

News and Comment

AMERICAN GIFT TO THE SOCIETÈ DE NEUROLOGIE

At a recent meeting of the Société de Neurologie de Paris, according to *The Journal of the American Medical Association,* the secretary-general read a letter from Dr. Hugh T. Patrick, Chicago, a foreign corresponding member of the society, in which the writer expressed his admiration for the work of French neurologists during the war and enclosed a check for 10,000 francs to help the society in continuing the publication of its transactions. The society unanimously voted its thanks to Dr. Patrick for his aid to the diffusion of its works, and expressed the hope that he, together with the new American members, would attend the next annual Réunion neurologique at which collaboration could be established between neurologists of France and the United States. This reunion will be held July 9-10. Only one subject will be discussed: "The Clinical Forms and Treatment of Syphilis of the Nervous System." The titles of discussions and the names of those who anticipate being present should be communicated to the secretary-general, Dr. Henry Meige, 55 Rue de Grenelle, Paris.

Abstracts from Current Literature

LE SINERGIE RIFLESSE SPINALI (SYNERGIC SPINAL REFLEXES). OBSERVATIONS ON SPINAL CORD INJURIES. Lodovico Gatti, Riv. di patol. nerv. **24**:165 (Sept. 13) 1919.

This extensive, well written article of fifty-five pages is divided into six chapters. The bibliography is large. It is carefully abstracted so as to make it available in English.

1. *History of the Physiology of Spinal Reflexes.*—The first chapter deals with this subject and is an extensive review of the literature. The first knowledge of the automatic functions of the spinal cord is due to the discoveries of Prochaska in 1784. Next come Vulpian and Brown Séquard and until recently these studies were limited to the physiologic laboratories and resulted from experiments on animals, the most important of which were done by Sherrington. These experiments, however, were brought into the field of clinical medicine by Pierre Marie. Sherrington's experiments on the cat illustrating decerebrate rigidity in the prepared animal are not influenced by section of the posterior columns of the cord, relaxation only being induced by section of the lateral columns below the decussation of the pyramids. Mention is also made of the fact that the rigidity involves only the extensors of the body, the flexors being only mildly tonic, the tonicity not being influenced by cord section. Excision of the cerebellum and the labyrinths, while diminishing the rigidity, does not abolish it. The rigidity is not abolished even if all the nerves of the skin and the other muscles except the extensors are cut; only when the nerve to a muscle itself is cut does relaxation take place, or when the posterior roots supplying that muscle are severed. This tonic influence is attributed by Weed to the red nucleus. Sherrington's observation that the tonicity is limited to posture maintaining muscles only is recalled. This is the postural or static reflex.

This static reflex is found to apply inversely, however, to the flexors as shown by Brondgeest in the spinal frog whose erect attitude is maintained by flexors. Sherrington's law of contraction of the posture maintaining groups and relaxation of the antagonists is discussed in relation to the static reflex. The work of Magnus and Kleyn support these views. Flexion of the head of the decerebrate cat decreases the tone of the extensors of the forelimbs and increases the tone of these in the hind limbs, whereas in bending the cat's head backward the reverse is true. The authors attributed this to stimulation of the proprioceptive nerves of the neck and of the vestibular mechanism, as destruction of the labyrinths and immobilization of the neck or cutting of the cervical roots have demonstrated. These authors also noted the unilateral head and trunkal synergy depending on these facts in the prepared cat. These facts have been corroborated by P. Marie in the hemiplegic patient and by Magnus and von Kleyn in normal men. When the head is inclined to 45 degrees below the horizontal level (the subject lying flat) the extensors of the legs show tonic increase which is decreased when the head is turned 160 degrees from this position. This can be demonstrated less actively, however, in children up to 3½ months old. In the prepared cat the static reflexes are inexhaustible and these muscles show the same tonic status whether they are lengthened or shortened in the act of maintaining posture.

According to Sherrington, therefore, there are two systems innervating muscle, one the static reflex residing in the midbrain and the other the flexor having essentially a phasic action mainly of protection, which is purely an inherent spinal cord function. The work of Bayliss, Uexküll, Parnas and Sherrington seems to show that there are actually two groups in the musculature of animals, one of phasic movement and the other static. These authors demonstrated this fact chiefly in bivalves. In vertebrates, however, purely static muscles are not found except possibly an analogous group in the smooth sphincters. In vertebrates, Botazzi assigns this dual rôle to the musculature, the sarcostiles being the contractile element and the sacroplasm the tonic. Perroncito and Boecke, Mosso, Van Boer and Langeaan have discovered a double innervation of striated muscle, one from the anterior horn cells and the other from sympathetic nonmyelinated fibers probably having to do with tonic impulses.

The writer calls attention to the works of Negrin, Lopez and von Bruecke who found that extirpation of the abdominal sympathetic does not produce a lasting atonia of the musculature of the same side. All these experiments unite to show that while decerebration reveals an automatic static tonic mechanism, this cannot be compared to the tonus of voluntarily contracting muscles, which exist in all normal persons. These static reflexes have also been demonstrated in normal cats and in man.

The author made his study because of the meager records of static reflexes in man of mesencephalic origin. In order to differentiate these from the phasic reflexes, he reviews our knowledge of the latter. For the phasic reflex studies the spinal animal must be used, despite the fact that in decerebrate animals the extensor hypertonus overshadows the phasic flexor system. In spinal animals, therefore, a painful stimulus applied to the hind legs causes a prompt retraction in all of their segments; the flexion reflex of Sherrington. Any part of the leg is a reflex zone except the nearest proximal portion to the stimulus which often shows a homolateral extension reflex, or the contralateral limb will show extension—the crossed extension reflex. If instead of nociceptive, mild stimuli are used, rhythmical reflex phenomena are demonstrated; in addition to the flexion reflex in the stimulated limb, one sees a crossed extension reflex or a flexion reflex of the contralateral foreleg with extension of the homolateral limb. With the limbs in this position the contraction formula now becomes inverted into a series of rhythmic movements reminiscent of the walking movements; the walking or running reflexes. Nociceptive stimuli inhibit these, but if such a stimulus is applied producing the flexion reflex and continued, the walking or running reflexes may be produced in the contralateral limbs. The rhythmic reflexes can also be produced by stimulating the cord itself electrically in the spinal dog. The scratch reflex can also be produced by stimulating the skin of the back and neck, the hind leg of the same side being used in rhythmic attempts to brush away the stimulus.

2. *The Synergic Pathologic Reflexes in Man.*—The author prefers with Kroll and Biondi to call these synergic reflexes rather than by any interpretative name such as Babinski's term "reflexes de defense" or Marie's "reflexes d'automatisme medullaire." In ordinary spastic paraplegias a painful stimulus applied to the foot produces the flexion reflex of the spinal animal. This is best obtained by forced passive plantar flexion of the toes

(P. Marie). Where this appears spontaneously, however, Babinski believes it is due to visceral stimulation. It has been known to occur during defecation, and Marinesco and Noica have seen it follow forced inspiration and coughing. Normally the movement of retraction of the limb with plantar flexion of the toes occurs in response to painful stimuli in man, and Babinski holds that obtaining this response by stimulating any part of the limb except the plantar surface, is pathologic, providing one gets besides flexion of the limb at all three segments, dorsoflexion of the big toe.

The Sensation of the Flexion Reflex in Motor and Sensory Paraplegics: Claude noted the subjective sensation which accompanied this movement in absolutely anesthetic paraplegic patients. The sensation is referred to the stomach and accompanied by a grimace. The author noted that this occurred only in those cases in which the flexion reflex causes trunkal motion. It was noted in patients with the anesthetic sensory levels not much higher than the umbilicus, in patients with anesthesia to the tenth dorsal vertebra, and only when the stimulus was painful enough to cause abdominal contraction. The factor of compression of the diaphragm caused by compression of the visceral mass enters into this, aside from possible obscure visceral sensory factors.

Extension Reflex: This is much rarer than the flexion reflex. All segments of the limb extend with plantar flexion of the toes. The reflexogenous zone may extend to the perineum or the loin or higher but in these cases the thigh may belong to a reflexogenous zone giving the flexion reflex instead of the extensor.

Crossed Reflexes: These are as described above, except that they are seen in the contralateral limb. Most commonly one sees the flexion reflex on one side with plantar flexion of the toes and foot of the opposite foot. This must not be interpreted as a crossed plantar flexion but as an abortive crossed extension reflex. At times, Walshe and Sherrington have noted the flexion rebound after a crossed extension reflex. Dorsiflexion of the big toe, however, is part of this rebound. The author believes that this is caused by the expansion of the flexor muscles in the extensor thrust causing a reflex in flexion by rebound. The inhibition of antagonists is illustrated in the extension reflex and is illustrated further in the study of clonus. With patellar clonus, the limb being placed in flexion, the clonus is increased in the opposite knee and diminished in the flexed limb.

Rhythmic Reflexes: The author has never seen them in his cases. Marie obtained a homolateral alternating flexion extension reflex by faradic stimulation of the skin of the inner thigh and a contralateral one by provoking a flexion reflex.

3. *Babinski's Sign and the Flexion Reflex.*—The regularity with which the triple retraction of the limb occurs with dorsiflexion of the big toe has led Marie and Foix to say that the Babinski reflex is part of the flexion reflex to which Babinski objects. Marie believes that the triple reflex retraction and the Babinski sign are automatic movements seen normally in the walking biped, and naturally plantar flexion of the toes occurs during the extension reflex because in walking when the limb is extended, the plantar flexors contract to clutch the ground and thus aid progression. According to Marie, the Babinski response is the minimum response to plantar excitation, a stronger stimulus being necessary for a flexion reflex response. The author describes two cases in which the Babinski response coincided with a lengthening of the limb giving an appearance of synergy between these two movements. He

says that he dealt in these cases not with an extension reflex because every stimulus increased the extensor spasm, and that this spasticity opposed the usual reflex because the extensors contracted more strongly. The dorsiflexors, while normally stronger, became less dominant because in the extreme extension of the limb due to the spastic paraplegia the points of their insertion were lengthened while the plantar flexors shortened theirs and made this movement mechanically easier.

The author agrees with Walshe that the Babinski is not the minimum motor response to plantar stimulation because by palpation the flexors of the leg were felt to contract more strongly in this test. That the dorsiflexion of the toe is more evident is due to the fact that the extensor longus hallucis has only to move the big toe while the flexors of the leg have a larger task. This contradicts Marie's views that the Babinski represents a minimum motor response to reflex stimuli. The author thinks that it is simply an expression of the reflex activity of the muscular group which is typographically the lowest in the flexor system. He relates a case in which a few days after injury the Babinski sign occurred alone and only on one side with mild contraction of the hamstrings. For a few days all voluntary movements, except that of the extensor hallucis, had disappeared in the extremities. While it is difficult to discover why all except this one muscle were paralyzed, the fact remains that the complete paralysis and areflexia showed that apparently a general inhibition of all spinal motor apparatus existed except in the extensor hallucis, making it possible to observe the flexion reflex essentially localized to this one muscle. The Babinski response does not accompany the flexion reflex only in rare cases, and some of these are caused by contractures in plantar flexion of the toes due to the weight of the bed clothes causing a pressure ankylosis and contractures, thus making dorsiflexion of the big toe impossible. As Walshe points out, the same thing may happen when there is contracture of the extensor hallucis less marked than in the proximal muscles of the leg, the marked dorsiflexion of the foot masking the mild dorsiflexion of the big toe. The author then cites Cases 5, 6 and 7 as examples of dissociation in the flexion reflex due to disturbances of nutrition in the cord because of infection and trauma. He believes that the Babinski sign was not obtained because the response was not a true flexion reflex, the movement occurring in the adductors of the leg. The reflexogenous zone was not in the foot as is customary with the flexion reflex but in the inner side of the thigh, the genitals and the perineum. In these patients even a light stroke of the pin caused a sharp adduction of both adductors and the glutei and retraction of the arms. These were the only muscles to respond. The author believes that here we deal with a true closure reflex such as would normally oppose the entrance of anything near the perineum, and not an incomplete flexion reflex.

Clinical Experiences with the Babinski Phenomenon: When the leg is flexed on the thigh, the patient lying face downward, the Babinski sign will often be substituted by plantar flexion of the toes; but this occurs only when the Babinski phenomenon is present to a moderate degree, for where this sign was marked and other synergic reflexes were present the author could not make the Babinski reflex disappear by placing the patient in the above attitude. In assuming this position there is a relaxation of the flexors and a tautness of the extensors of the leg, those of the foot not being influenced. The author, by overflexing the leg on the thigh in the prone position, was often able to cause the Babinski reflex to disappear and reappear by extending the leg a little. He points out that these movements are practically the only function of the lower extremity in walking in contrast to the varied functions of the hand and fingers. Necessarily, therefore, there must be an intimate synergic association between the flexor and extensor groups of cells in the cord for the performance of this automatic function, analogous to the system of oculogyric and cephalogyric movements. So one finds that the rectus femoris muscle in the superior portion is part of the flexor system while the remainder is an extensor. The lower end of the quadriceps, the gluteus maximus and the posterior muscles of the legs are extensors, but the psoas, anterior rectus, sartorius, gracilus, pectineus, the adductors and the tensor fasciae, the hamstrings, tibialus anticus and the extensors of the toes are the flexors, according to this grouping. There is a tonus synergy between these groups clinically proved by the fact that stimulating the (Babinski test) flexor system inhibits the extensor groups (patellar clonus). The author, experimenting on paraplegics with complete anesthesia to eliminate the painful element of the stimulus, was able to show in nineteen out of twenty-one paraplegic soldiers that causing a strong extensor contraction by faradization of the anterior crural nerve, a strong faradic stimulation of the plantar surface of the foot failed to produce an extensor response of the big toe (Babinski sign). If instead of the anterior crural the obturator was stimulated, the Babinski reflex was obtained. Strong currents must be used on the nerve and not the muscles to obtain this phenomenon. This the author feels shows the intimate connection of the Babinski reflex with the flexor group, as does also the fact that often passive extension of the foot on the leg will cause the extensor big toe response as will also the mere raising of the heel above the bed passively. In these movements the stimulus caused by stretching the posterior leg and thigh muscles is enough to produce a flexor response in all three segments of the extremity if the paralysis is profound enough (automatism of cord) but in milder cases the response of the extensor hallucis is obtained (Babinski sign) alone as part of the flexor response. In testing these points on patients the author warns that too large an area must not be exposed to stimulus because in certain cases the Babinski sign may be elicited by pinching the cheek or the ear. The patients should rest on a firm, flat bed, all points of contact with the body removed (bed clothes), the room should be warm and care must be taken in the passive muscular movements not to stroke or otherwise irritate the skin. The organic reflexes of the patients must also be discounted for a distended bladder or rectum will often cause the Babinski response independently of any other stimulus.

4. *Physiologic Significance.* — The author believes that the flexion reflex has not the same significance as the rhythmic reflex (running) because a single painful stimulus produces the first while continued mild or painful stimuli are necessary to bring out the latter. Marie does not believe the flexion reflex is a defense reflex but the author objects to Marie and Foix's views that it is an expression of "spinal stepping," i. e., a phase of gait more particularly because if this were true then extension of the limb should precede flexion as occurs normally in animals. As the plantar surface of the paw touches the ground, the toes are in plantar flexion but the limb is in extension. The author admits that some examples of initial rapid extension of the limb occur before the flexor response in man or only a plantar flexion of the toe precedes the flexion reflex with dorsiflexion of the toes, but usually the response is suddenly flexor alone and cannot be a synergic walking phasic response to plantar stimulation. Babinski, Walshe, von Woerkom and others,

however, think the flexion reflex is purely defensive. They admit the difficulty of explaining it as a defense reaction when provoked by stroking the chest or abdomen, but they say the same objection holds good for those who insist it is a walking reflex. They insist that no matter how wide the reflex zone may be the plantar surface of the foot has always the lowest threshold for the stimulus, therefore the flexion response always has a certain protective quality. The author does not believe either explanation is complete. He feels that the stimulus arriving in the anterior horn cells of the automatic spinal animal diffuses through various groups of cells awakening an automatic synergic response which may be a habitual response to serve any one of several ends. There is no specificity of purpose about the response, it is only a movement. The animal flexes his limb in walking and in withdrawing it from painful contact and the child in his first days does the same purposelessly. The fetus assumes this position. In other words, if the rhythmic reflexes are ambulatory responses, the closure reflexes defensive, the synergy between contraction of the bulbocavernosis and flexion of the legs is a sexual reflex; the retraction of the lower extremity represents, therefore, not an action but a phase in the completion of one, which may be the running reflex synergy or something else.

Regarding the Babinski response, it is necessary to say that the upper extremity has many movements of cerebral origin, the lower being almost wholly limited to automatic extensor and flexor movements at the three joints comparable to what is seen in the animal; therefore these movements, when the control of the cerebrum is cut off, remain as the automatic function of the spinal cord as determined by habit and phylogenesis. In man, however, the development of the cerebrum has reached such a state that the automatic synergic reflexes in the cord cannot be compared in extent and number to those found in the spinal animal. The author who has observed many hundreds of spinal cord injuries as a result of the war, has never seen the rhythmic reflex (running reflex) and only rarely the crossed extension reflex.

5. *Cutaneous Reflexes: The Bulbocavernous and Sexual Reflex.*—The cutaneous reflexes are analogous to the synergic and appear more as reflex movements adapted to defense. These reflexes are not cortical, however, for transverse lesions of the cord have been described with their retention although, as Sherrington has shown, the cortex inhibits them. The cutaneous reflexes are spinal and have a phylogenetic habitual significance. The author looked for Onanov's sign in all his cases. It is obtained by stimulating the skin of the scrotum or the penis, the bulbo and ischio cavernosi being felt to contract in the perineum (S3) and accompanying this other muscles, chiefly the levator and sphincter ani, contract. Usually this reflex disappears with impotence due to old age, tabes, etc. The author found it persisted in severely injured soldiers, even when there was complete paraplegia with anesthesia and retention or incontinence, providing the prepuce and glans were sufficiently stimulated. It is not a priapism due to vasomotor paralytic stasis in the corpi cavernosi, such as is seen in some complete transverse lesions of the cord, but a real erectile contraction. In one nearly complete severance at the sixth dorsal level (operation) with paraplegia, anesthesia, bed sores, flexion reflex with Babinski sign (bilateral) areflexia and incontinence, the bulbocavernous reflex was obtained even by pricking the skin of the anus, perineum and gluteal region. In many cases this reflex was accompanied by contraction of the biceps (flexion and rotation of the limb and eversion of the leg). In only two of 150 cases showing the bulbocavernous reflex was this contraction of

the biceps absent, and when the biceps contracted strongly the semitendinosis and semimembranosis also often contracted. This, however, is not a pathologic reflex because it was found present in 60 per cent. of normal soldiers of from 20 to 30 years of age. However, in the normal man the response is less active, the stimulus must be frequently repeated and is easily exhausted and the contraction of the other hamstrings is never seen. A unilateral absence of this reflex is seen in sciatica. In spinal hemiplegia the reflex may be unilaterally present. Cases 8, 9 and 10 illustrate these points. That it is often the only reflex obtainable besides the flexion reflex is illustrated by Cases 11, 12 and 13. The author points out that the bulbocavernosis (S3) and the biceps (L4 and S1) are not segmentally or peripherally connected; therefore it must be a synergy and related to the cutaneous reflexes with their limited brief responses rather than to the ample widely moving synergic reflexes so-called, previously described.

The writer found that by pressing downward on the clitoris in forty out of seventy women (57 per cent.) a contraction of the vulva (bulbo cavernosis) and of the sphincter and levator ani, with synergic bilateral contraction of the biceps occurred. The clitoris and penis therefore are not only homologous structurally but have the same reflex nervous mechanism.

The analogue of the bulbocavernous reflex in man is found in the spinal male frog killed during mating time. Touching the animal's chest causes a movement of embracing (Rana and Bufo). The author believes it is a coitus synergic reflex, because in animals having no glutei and who do not assume the erect posture the flexors of the leg are the muscles used in coitus for fixation, the muscles above the pelvis being used to introduce the penis and complete the act. In man the rotation of the leg on the pelvis is slight, the glutei acting more strongly to rotate the thighs on the pelvis.

6. *Clinical Significances.* — In cord lesions involving mostly the pyramidal tracts the contention of Walshe that there is a double innervation of the two systems—flexor and extensor—seems to be borne out; the extensor static system is stronger hence the patellar and ankle clonus and hyperflexion, but increase of the synergic reflexes also occurs due to flexor hypertonus. The extensor hypertonus is static and contracture occurs in extension through its dominance (decerebrate rigidity). In these purely pyramidal injuries the flexion, phasic musculature causes only mild flexion reflexes. When, however, the lesion is complete, the paraplegia should show flexor responses because the paracerebellar tonic impulses do not reach the extensor system allowing the purely spinal flexor system full play, resulting in some cases in contractures in flexion which may be permanent.

The author saw only one case of complete areflexia of all types. This occurred in a low complete lumbosacral crush (Case 14). The greatest signs of automatism of the cord were found in partial lesions, without too much trauma, infection or circulatory disturbances in the cord. The presence of synergic reflexes is a sign of grave cord injury. The author never saw a patient recover or improve much who had these signs. An apparent contradiction as to the prognostic seriousness of these signs is found in cases of Friedreich's and heredocerebellar ataxia in which sometimes the reflexogenous zone includes the whole body without the presence of the serious paralyses or contractures seen in paraplegic soldiers.

These reflex phenomena begin in the second week after injury, increase in intensity for a while then remain stationary (in one case for two years) for a long time. They disappear sometimes in complete lesions.

Babinski's flexion paraplegia is not, according to Walshe and Thomas and the author, an indication of mild pyramidal involvement, but is due to the frequent incitement of the flexion reflex, the lower extremities assuming this attitude as a result of contractures. At first, the author insists that in these patients with flexion paraplegia, another reason than the mechanical contractures is operative, for, after the initial shock was over, these patients lying in passive extension (flaccid paralysis), soon changed to active spastic flexion. The paralysis is probably originally one in flexion, the later attitude being inhibited by the shock. All of the author's patients with flexion paraplegia were seriously injured, with complete motor and sensory loss. In these cases the ankle and patellars are diminished and the biceps and other flexors give active reflex responses.

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UEBER DIE FASERANATOMISCHEN BEZIEHUNGEN ZWISCHEN DEN KERNEN DES THALAMUS OPTICUS UND DEN FRONTALEN WINDUNGEN (FRONTAL REGION) DES MENSCHEN (ANATOMIC RELATIONS OF THE NUCLEI OF THE OPTIC THALAMUS AND THE FRONTAL REGION OF THE HUMAN BRAIN). Tsunesuke Fukuda, Schweiz. Arch. f. Neurol u. Psychiat. **5**:325, 1919.

The investigation undertaken by Fukuda was for the purpose of determining whether the frontal lobe is provided with a more or less definite projection system or whether this is absent as maintained by Flechsig; also whether definite areas of the frontal lobe are intimately related with given portions of the thalamus. He resorted to the method of secondary degeneration as produced by old, more or less sharply demarcated lesions of the frontal cortex. Thirteen brains were studied in great detail.

The first patient, 72 years of age, who died of carcinoma of the esophagus, had attempted suicide fifteen years previously by shooting himself in the right frontal portion of the head. Investigation showed that the injury was located in the first, second and third right frontal convolutions, involving principally the posterior third, a portion of the operculum and the anterior portion of the first temporal convolution. There was a well demarcated secondary degeneration in the dorsal lenticulo-optic segment of the internal capsule. Evidence of progressive secondary degeneration of the nerve cells was noted in the nucleus lateralis, the nucleus medialis and the nucleus ventralis anterior of the thalamus.

The second case was that of a woman, 59 years of age, who presented signs of brain tumor. She was operated on, and it was ultimately proved that the condition was encephalitis with destruction of the second and a portion of the third frontal convolutions. A secondary degeneration was found in the nucleus lateralis, the anterior portion of the nucleus medialis a, and a slight diffuse degeneration in the anterior portion of the nucleus ventralis anterior.

The third brain was that of a man, 50 years of age, in whom a dementia paralytica was suspected by the mental symptoms, but who proved to have a sarcoma about 5 cm. in diameter in the anterior portion of the left frontal lobe. A portion of the first and second frontal convolutions was destroyed with secondary degeneration in the nucleus medialis a, the anterior portion of the nucleus lateralis, and the nucleus ventralis anterior.

The fourth case was that of a 5 year old microcephalic idiot. Necropsy showed a marked and rather extensive cystic condition of the brain. On the left side, only the inferior convolution of the frontal lobe, the island of Reil, the greater portion of the temporal lobe, the caudal portion of the gyrus marginalis, and the ventral half of the occipital lobe of the left hemisphere remained. On the right side, the basal portion of the frontal lobe, the caudal portion of the second and third frontal gyri, the temporal lobe, and the basal portion of the occipital lobe remained. On the left there was evidence of secondary cellular degeneration in the anterior portion of the tubercle, the lateral nucleus, the nucleus ventralis anterior, the nucleus medialis a, and the nucleus ventralis a and b. On the right, the anterior half of the anterior tubercle showed slight degeneration, the nucleus lateralis complete degeneration, and the nucleus medialis marked degeneration.

Case 5 was a 13 year old child with encephalitis involving the caudal portion of the second and third left frontal convolutions and the anterior portion of the pars opercularis of the precentral convolution. Secondary degeneration could be followed through the internal capsule into the thalamus, which disclosed a moderate degeneration in the nucleus ventralis anterior and the central portion of the nucleus lateralis; the nucleus medialis a was degenerated somewhat more markedly, while the nucleus medialis b contained only a few degenerated cells.

Case 6 was a patient 47 years of age, who had had an extensive hemorrhage of traumatic origin into the left frontal lobe, followed by the formation of a large cyst. The ventromedial portion of the first frontal, including the gyrus rectus, and a portion of the posterior medio-orbital region remained. The anterior part of the left internal capsule was markedly destroyed. There was a marked degeneration of the nucleus medialis a, particularly in the anterior portion, the nucleus medialis b, the nucleus lateralis, and the nucleus ventralis. The tuberculum anterior was reduced only a trifle in volume.

The seventh case was that of a 54 year old man who died of a glioma located in the left island and spreading into the third frontal convolution. A second, smaller glioma was noted in the extreme anterior portion of the lenticulostriate body, involving the pyramid. Secondary degeneration was noted in the anterior third of the nucleus ventralis anterior and in a small portion of the nucleus medialis a.

Case 8 had a tubercle about 2 cm. in diameter located in the posterior portion of the left first frontal convolution and the paracentral lobe. From this area degenerated fibers could be traced through the medial portion of the internal capsule to the thalamus. The anterior tubercle, the nucleus medialis a, the nucleus lateralis, and the nucleus ventralis anterior showed degeneration.

The ninth case was that of a man, 37 years of age, afflicted with endocarditis, who had had an apoplectiform insult nine months before. Necropsy showed a multilocular cyst in the second and third frontal convolutions and in the temporal lobe. Practically all of the thalamic nuclei, with exception of the tuberculum anterior, showed high grade degeneration. The latter was normal throughout.

Case 10 was that of a man, 55 years of age, who died of pulmonary embolism and who had sustained an apoplectiform insult three years before. A lesion involving the inferior two thirds of the precentral gyrus and the caudal portion of the third frontal was found. Secondary degeneration proceeded through the internal capsule into the thalamus with degeneration in the nucleus ventralis anterior, the dorsal third of the nucleus lateralis and the nucleus medialis b.

Case 11 was that of a man, 75 years of age, who had a hemorrhagic cyst in the median portion of the second and third convolution, from which a degenerated bundle of fibers could be traced through the internal capsule to the thalamus with destruction of the nucleus lateralis, the nucleus ventralis anterior, and the nucleus medialis b.

Case 12 was that of a patient 44 years of age, who on necropsy showed a sarcoma involving the upper half of the left central convolution extending to the medial surface of the hemisphere. There was a slight degeneration of the internal capsule. The middle section of the nucleus medialis was destroyed.

Case 13, a patient 35 years of age, had a sarcoma located in the anterior portion of the first and second frontal convolution on the right side. The nucleus ventralis anterior and the nucleus lateralis were degenerated.

In his summary, Fukuda states that, according to Flechsig, the massive bundle in the anterior portion of the internal capsule proceeds to the frontal lobe. The latter assumed, however, that these fibers change their course in the region of the knee of the internal capsule and, making an arch, break through the pyramidal tract and ascend to reach the anterior portion of the sensory area. He looked on the frontal lobe and these tracts as being a large association center which furnishes the anatomic basis of intellect.

Fukuda constantly found evidence of secondary degeneration proceeding from the corresponding fibers of the internal capsule to the lateral and anteroventral portions of the thalamus. The relations of the anterior tubercle and nucleus medialis are not so constant.

While it was established that in larger defects of the frontal lobe, the anterior portions of the thalamus, particularly the nucleus lateralis, undergo degeneration, the more exact location of lesions in the frontal lobe, which bring about degeneration in so many portions of the thalamus, becomes more difficult. This is particularly true of this series in which the location, the extent, the age of the patient, the duration of the lesion and the nature of the pathologic processes were so variable. In five cases tumors were present, in one each porencephaly and embolism of the Sylvian artery, in two the lesions were traumatic, in four encephalitic. On the whole, therefore, the lesions in the author's cases showed little tendency to be sharply demarcated.

His conclusions relative to this relationship are: The posterior portion of the second frontal convolution and the nucleus medialis a, following von Monakow's division of the median portion of the thalamus, are intimately related.

The cortical representation of nucleus medialis b is the posterior portion of the third frontal convolution.

The cortical representation of the nucleus lateralis is not sharply demarcated, but seems to be widely distributed over the frontal lobe.

Degeneration of the tuberculum anterius is, so far as the frontal lobe is concerned, only a relative affair. In no single case was the entire nucleus degenerated. The author feels, therefore, that the tuberculum anterius assumes a peculiar relationship, which may in part be explained by its participation in the relation with the corpus mamillare through the bundle of Vicq d'Azyr.

The nucleus ventralis anterior and the nucleus dorsolateralis show the same general changes in frontal lobe lesions in that the small nerve cells, particularly in large lesions of the frontal lobe, are almost totally destroyed. The cortical representation of these two nuclei must be an extensive one.

In general it may be said that the median portions of the thalamus are less degenerated than the lateral, the degeneration becoming less the nearer the ventricle is approached. The author infers that the thalamocortical projection fibers from median portions of the thalamus become progressively more divergent, as opposed to those arising from the lateral portions; the median portion does not degenerate nearly so much as the lateral and only in part when the posterior portions of the second and third frontal convolutions are involved. His observations do not confirm those of Nissl, who by experimentally removing the subcortical white matter found degeneration of all nerve cells in the right thalamus as far as the commissura mollis, that is, inclusive of the central gray matter. This does not occur in man. On the contrary, Fukuda found that, even in extensive lesions, the gray matter about the third ventricle, the tuber cinerium and the majority of fibers in the tuberculum anterius, remained practically intact. In serial sections of a cat's brain from which von Monakow had removed the entire cortex, he found a complete degeneration of these cells in only one instance, which he infers was not the result of secondary degeneration, but was due to a primary encephalomalasic necrosis. The cells, almost exclusively preserved, are small ones, and particularly those near the lumen of the third ventricle; these are not related to the fibers of the capsule which run to the frontal lobe. Experimental work of von Monakow shows that the chief cells of the thalamus are related to the cortex, while the smaller ones stand in intimate relation to the retrothalamic fibers coming from the tegmentum.

Woltman, Rochester, Minn.

SUR UN CAS D'ACROMEGALO-GIGANTISM AVEC PHYCHOSE MANIAQUE DEPRESSIVE (A CASE OF ACROMEGALIC GIGANTISM WITH MANIC DEPRESSIVE PSYCHOSIS). Parhon and Stocker, Rev. neurol. **35**:573 (July) 1919.

The authors refer briefly to the work of eminent writers whose results in studies of the thyroid, pituitary and thymus glands establish beyond question the influences of these glands on growth and development of the human organism. He then quotes Henri Meigs (Li gigantisme, 1912) who inquires whether one may not assert that the enfeeblement of the genital function plays the most important part in the occurrence of skeletal anomalies.

Not only do physical anomalies show themselves as the result of disturbances of the internal secretions, but there is a close relationship between psychic disorders and disorders of the endocrine system.

The authors refer briefly to the Abderhalden reaction, especially in dementia praecox and in manic depressive cases, and then list under eight heads phenomena common to Basedow's disease and the circular type of manic depressive insanity to show how close is the relationship between them:

1. Clinical symptoms of thyroid disturbances in manic-depressive cases, such as exophthalmia, goiter, tachycardia, etc.
2. Resemblance in the mental state of those suffering from manic-depressive insanity and from Basedow's disease.
3. Basedow's disease, and an affective psychosis in the same patient.
4. The average or medium weight of the thyroid body in the manic-depressive patient is equal to the maximal or upper limit of that in normal persons.

5. The frequency of the two disorders in the female sex.

6. The coincidence of the two diseases at periods in which hyperthyroidism is common, such as at puberty and the menopause.

7. Hyperexcitability of the vegetative nervous system in both disorders.

8. The therapeutic effect of thyroidectomy.

According to Brunet (*These de Paris,* 1899), mental troubles are eight times as frequent among acromegalic people as among normal persons. Reports of many cases of acromegaly with frank psychoses have been published. In the majority of cases it is the manic, or the depressive phase of manic-depressive insanity.

The authors cite authorities who have reported one form or another of thyroid disturbance occurring in cases of acromegaly, and other authorities who have tried to show that in cases of gigantism elementary psychic disturbances are the rule, such as infantile or puerile reactions, effeminate traits, lack of energy, lack of will, etc.

The weight of the thyroid in giants is usually greatly increased; cases in which the weight was as high as 75 and 112 gm. have been reported.

The true psychoses are not frequently found among acromegalic patients. Cromwell's porter, Daniel, had a mania for prophecy. Parhon (1910) describes a case of gigantism with periodic psychosis and Gelma (1912) reported a case of Basedow's disease combined with gigantism and a periodic psychosis.

The authors report in detail their own case of acromegalogigantism complicated with a periodic psychosis. The more interesting features in the case are:

1. The patient was a man, 30 years of age, single, a bank clerk.

2. He was first seen at the clinic for nervous diseases at Jassy, in May, 1916. He was transferred to the hospital for the insane at Socola in June of the same year, and discharged the following September. He was readmitted on March 28, 1918, in manic excitement, having delusions of persecution.

3. Family History: His father was tall, high strung, impulsive, not alcoholic or syphilitic. His mother died of pulmonary tuberculosis. He had two sisters—the older normal, the younger a congenital deaf mute.

4. Makeup: He was quiet and amiable as a child; he became malicious and ill-tempered; later he was again even tempered and congenital. Alcoholism and syphilis were denied up to the age of 10. At that age his growth was about normal; from then on, growth was unusually rapid. At 12 he had the appearance of a youth of 15.

5. Physical Examination: The patient was 6 feet tall and thin. At the age of 16 or 17 he was obese. His head was small compared with his height. The frontal sinuses were prominent, giving the appearance of enophthalmos. The iris of the right eye was gray; of the left, brown. His tongue was thick (6 cm. wide). There was striking prognathism of both maxillae; the malars were prominent. The ears were flaring but without other stigmas. His face in general was symmetrical, but developed out of proportion to the rest of the head. The thyroid gland was negative clinically.

The larynx and the pelvis were feminine in type The genital organs were small, but not otherwise abnormal. Sensibility and motility were negative.

6. Mental examination revealed an anxious appearance, wringing of the hands, a depressed and sad mood and frequent deep sighs. The voice was deep and whining. His gaze was fixed, and he was indifferent to his surroundings.

He was introspective; his speech was confused; and at times he acted as if recalled from another world. Replies to questions were labored and retarded.

He had delusions of unworthiness, including ideas of being sacriligious, of practicing onanism and adultery.

(Testicular extract seems to have had some beneficial effect.)

During his second commitment he was quiet and respectful, but mingled with other patients, and engaged in spontaneous conversation.

His attitude was that of extreme submission. When the physician made his rounds the patient would fall to his knees, and embrace the physician's hands and feet. He asked for prayers of others. He refused food and drink. He had grandiose ideas about his parents whom he spoke of as though deified. At the death of his mother all hope left him.

He frequently wrote letters of supplication to the director of the hospital and in them reiterated his ideas of self-depreciation. Opposed to such ideas, he frequently exaggerated his own abilities as a child and student. He wished to complete his studies that he might bounteously bestow beauty and good on the world. For this reason he begged to be released, that he might go on with his doctorate. At times he expressed some insight into his mental illness, and expressed philosophical ideas about the work of such poets as Goethe and Shakespeare and quoted Schopenhauer. Then he would drop back into his former state, promising to become a "normal man" if set at liberty. He spoke of his life of chastity, and for this reason was determined to marry a princess; but he was sure that a queen had had relations with him; that was why he was being persecuted.

On the following day he would deny all these fantasies. Two months after admission the patient was discharged almost recovered.

Authors' Conclusions.—1. The authors do not believe that the case presents simply a coincidence of mental disorders and acromegalogigantism.

2. They regard the case as illustrating the dependence of psychic phenomena on physical disorder.

3. The influence of the internal secretions is as great in gigantism as it is in acromegaly.

4. One internal gland cannot be altered in function without a disfunction of the other internal glands.

5. Manic-depressive insanity may in certain cases be due to thyroid disfunction.

6. The authors' case shows how disorder of thyroid activity upsets the equilibrium of the other internal glands in an acromegalic giant and that the psychic manifestations of the manic-depressive type are apparently due to a functional disorder of the thyroid glands.

Jones, Detroit.

APHASIE ET APRAXIE (APHASIA AND APRAXIA). Laignel-Lavastine, Bull. méd. Par. **34**:277 (March 20) 1920.

The published report of a clinic by the writer gives with considerable conciseness his opinions regarding aphasia and apraxia.

For the former he studies spontaneous speech, the speech of repetition, reading aloud, spontaneous writing, writing from copy, writing from dictation, comprehension of spoken and written words, and divides the aphasia into two groups according to whether there is any trouble of interior language. If there is, one speaks of intrinsic aphasia and if there is not, of extrinsic aphasia.

The best known test for this is that of Proust-Wernicke-Lichtheim-Déjerine. The patient is shown an object and asked to open and close the hand as often as there are syllables in the word. In intrinsic aphasia the patient does not know the number of syllables. This permits the separation of pure motor aphasias from the aphasias of Broca in which motor aphasia is united with troubles of internal language. The extrinsic aphasias are divided sometimes into four groups: (1) pure motor aphasias, (2) pure alexia, (3) agraphia, and (4) pure word deafness. Déjerine doubts the existence of a pure agraphia and P. Marie questions the occurrence of pure word deafness.

The intrinsic aphasias are divided into two groups: (1) the aphasias of Broca, characterized by the loss of ability to articulate, and (2) the aphasia of Wernicke characterized by the loss of comprehension. The aphasias of Broca have usually sensory-motor association, such as hemianopsis, hemiplegia and hemianesthesia, which are absent in general in the aphasias of Wernicke.

In addition to the sensory-motor association, in the aphasias one finds agnoso-aphraxic association.

Considering the sensory inflow and motor outlet and normal transmittory interior connection, there are three apraxias: (1) the ideational apraxia or apraxia of conception, (2) ideomotor apraxia or apraxia of transmission, and (3) motor apraxia, strictly speaking, or apraxia of execution.

To analyze these apraxic disturbances one employs tests of movements of the following type: simple, reflex, expressive, descriptive and transitive (the latter involving the handling of several objects). It often happens that apraxia (in a right-handed person) is greater in the right arm. This is accounted for by a residual slight hemiplegia causing a right motorapraxia in combination with a bilateral ideomotor apraxia. The patient with ideomotor apraxia recognizes objects, their name and their use. This eliminates ideatory apraxia. He understands the end in view but cannot imagine the kinetic processes which would permit him to arrive at it.

It is necessary to distinguish between apraxia and ataxia. The ataxic patient directs himself hesitatingly toward an end and his conception of the end is correct. When he proceeds slowly he completes the action, and when he modifies his action, it is to improve it. The apraxic patient does not improve by repetition. When he tires he becomes more and more maladroit and never attains his purpose.

The localization of the lesion in ideomotor apraxia is established. It is the result (in right handed persons) of a lesion located in the left parietal lobe, more especially in the left supramarginal gyrus. A lesion of the left cerebrum determines a bilateral apraxia, which predominates on the right. When (in a right-handed person) the apraxia predominates or exists alone on the left, there is also either a lesion of the corpus callosum or an associated lesion of the right cerebrum.

As regards the relation between aphasia and apraxia, aphasia is a particular case of apraxia. Ideatory apraxia corresponds to dyslogia, motor aphasia to anarthria and ideomotor apraxia to aphasia.

Visual agnosia corresponds to word blindness and auditory agnosia to word deafness. There is further a parallelism between jargonaphasia and paraphasia and the parapraxia which is the result of agnosia. Motor aphasias are only a species of apraxias.

Davis, New York.

TRAITEMENT DE CERTAINES FORMES DU VERTIGES DE MENIERE PAR LA TREPANATION DECOMPRESSIVE (TREATMENT OF CERTAIN FORMS OF MENIERE'S VERTIGO BY DECOMPRESSIVE OPERATION). Henri Eboulker, Rev. neurol. **26**:493 (June) 1919.

Since the pathologic conditions of the labyrinth have become better known, the term "Ménière's vertigo" has been abandoned by modern otology. Nevertheless, the author uses it in this discussion because it has remained in current usage in general practice and serves to designate nonsuppurating labyrinthine conditions without specifying their pathogeny.

Ménière's vertigo is a syndrome characterized by deafness, tinnitus, vertigo and vomiting. Labyrinthine conditions secondary to suppurative processes of the middle ear present the same symptomatology. They reach the posterior-inferior region of the skull through large lymphatic and vascular communications. They end either in the formation of suppurating foci or in different meningeal conditions. Besides these suppurating conditions, the author believes there are nonsuppurating processes in which inflammation misses the labyrinthine apparatus and produces a certain degree of hypertension of the posterior-inferior region which calls for decompressive measures. He reaches this conclusion through analysis of symptoms and therapeutic evidence.

This syndrome, outside of the purely labyrinthine symptoms—vertigo, tinnitus, deafness and vomiting—presents headache with occipital localization. The patients localize the headache perfectly at the back of the head, on the same side as the tinnitus and deafness. In cases of vertigo, besides headache, one may also localize a sore spot on pressure, at the posterior portion of the mastoid. Before examination, the patients complain of these pains and give the impression that they have an inflammatory complication of suppurative otitis. "I must have an abscess." This is the way in which they describe their sensations.

Intracranial hypertension, which the author believes real in certain cases of vertigo, reacts on the labyrinthitis which then appears like a true "labyrinthitis of stasis" in the syndrome of compression of the cranial cavity.

Babinski, long ago, showed the usefulness of lumbar puncture in the treatment of vertigo. All patients with vertigo do not improve under this treatment, but some improve immediately. The patient feels as if he were born again; the tinnitus and the vertigo decrease or disappear, the vomiting ceases. Often the symptoms reappear soon but again disappear at the next puncture.

How can this sudden improvement be explained if it is not through a quick decompression of the posterior-inferior region of the meningeal cavity, reacting on the intrapetrous and extrapetrous labyrinthine passages? The resemblance is better seen in the evolution of the symptoms of patients who are not benefited, or slightly benefited by these punctures, and who apparently are cured by decompressive trepanation.

It is customary to judge the degree of hypertension by the pressure of the cerebrospinal fluid; but in cases of vertigo, it is not rare for the fluid to flow drop by drop. However, that does not prevent the patient from improving for a time after each puncture. Sicaid and Pullain have shown "that there does not exist any exact accordance between the hypertension of the cerebrospinal fluid, verifiable by puncture, and the hypertension of the intracranial cerebrospinal fluid." In several cases of cerebral tumor, confirmed by necropsy, it has been noted that, in spite of clear signs of cerebral hypertension—violent headache, vomiting, papillary stasis—the hypertension has not been discoverable by lumbar puncture. The cerebrospinal fluid at different punctures flows out slowly—at times, drop by drop. M. Claude says "We cannot gain information on the intraspinal pressure by the one proof of the way in which the cerebrospinal fluid flows through the needle; we must have a manometric measurement. Great pressure is observed even when the liquid flows drop by drop." The author has personally observed some significant cases in which there was absence of papillary stasis and a flow of cerebral fluid drop by drop coexistent with an enormous intracranial pressure. He cites cases that have come under his observation. Claude's manometric puncture should be applied particularly to the study of these arrested hypertensions, which it can help to bring into evidence.

Dry labyrinthitis corresponds frequently, from the anatomic-pathologic point of view, to arteriosclerosis of the labyrinth, as Escot has demonstrated, and to general arteriosclerosis of the arterial system. Labyrinthine hypertensions are often arterial hypertensions. Patients afflicted with arterial hypertension exhibit signs which are connected with increase of the cerebrospinal fluid and which can simulate in all respects the picture of a cerebral tumor.

Sometimes alterations of the fundus, which are really papillary stasis, can produce the change, but arterial manifestations account for the increase of pressure of the cerebrospinal fluid as lumbar puncture quiets them in a remarkable way.

In cases of arterial hypertension with intense headache, M. Vaquez advises the systematic use of bleeding in addition to lumbar puncture. He has drawn as much as 60 c.c. of fluid and has never had an accident. The headache and nervous trouble have been considerably lessened.

The author states that he has accepted from the work of M. Castaigne his opinions on arterial hypertension and intracranial hypertension. They appear to him to throw light on the pathogeny and treatment of the forms of Ménière's vertigo, connected with circulatory and cerebral manifestations and considered in all cases as purely local conditions. This symptomatic triad—arterial hypertension, labyrinthine hypertension and cerebral hypertension—constitutes the links in one chain. As the local lesion has its point of departure in the internal ear, whose vascular and lymphatic passages end in the postero-inferior region of the cranium, it is natural that the cerebral manifestations should exist principally in the cerebellar cavity and that, by preference, decompression should affect the same region.

In all serious labyrinthine conditions, it is important to search for the most frequent causal affections. There should be examination of the urine for albuminuria and diabetes and an examination of the blood for syphilis. These causal affections eliminated, we should look for arterial hypertension and pressure of the cerebrospinal fluid. Hypertensive medication, bleeding and lumbar puncture should be used methodically. If these are unsuccessful, we are justified in considering decompressive trepanation.

The author advocates, in certain cases, craniectomy under a local anesthetic. He has frequently performed under local anesthesia all forms of surgery of the skull, brain, face and neck. He refers to an article by himself in which he has enumerated eleven craniectomies of the temporal, parietal and occipital regions. He gives in detail the technic of his procedure. He cites the cases of two women troubled with a serious form of Ménière's vertigo. Vertigo, vomiting and tinnitus made existence intolerable. All the usual medications had been tried without result. One of the patients, the first one operated on, said that she would make an attempt on her life if the author did not succeed in helping her. He resorted to decompressive trepanation and the patient was cured. The second one was also operated on and cured. He reported these cases in February, 1913, giving details of these and at least two other cases with similar results.

JONES, Detroit.

BRACHIAL BIRTH PALSY, A PSEUDOPARALYSIS OF SHOULDER JOINT ORIGIN.

T. TURNER THOMAS, Am. J. M. Sc. **154**:207 (Feb.) 1920. Author's Abstract.

From a considerable experience with traumatic brachial paralysis, including eighteen cases in adults with flail shoulder joint and forty-four cases of obstetrical paralysis, it is concluded that in most cases the paralysis is not permanent but slowly disappears completely. In a small number of adult cases the shoulder joint becomes flail and these patients develop a permanent paralysis with atrophy of the shoulder muscles of varying severity, the rest of the limb showing much improvement but never becoming normal. In all of the cases of obstetrical or brachial birth paralysis, in which there was serious crippling of the limb with muscle atrophy, as late as 5 or 6 years of age, there was found a posterior subluxation of the shoulder joint. Those without any abnormality of the shoulder joint at that age had recovered full power and function of the limb. From this kind of evidence it is argued that the basic causal lesion is in the shoulder joint and not in the brachial plexus. The paralysis is real in the early stages but is termed a pseudoparalysis here in the sense that it is only temporary and is probably not due to a serious nerve injury but to inclusion of the branches of the plexus in the axillary inflammation resulting from the joint injury.

There have been no case reports of obstetrical paralysis in adults because by that age the paralysis has disappeared, only a varying crippling of the limb from some residual shoulder joint distortion remaining in the most severe cases. The adult case on which Madame Klumpke based her study was one of total permanent, flaccid paralysis of motion and sensation, and did not belong to this group of cases.

Postmortem and operative findings recorded in the literature showed that the nerves were involved in a cicatricial tissue formation, probably resulting from their proximity to the articular and peri-articular tissues damaged at the time of the original injury. The reported nerve ruptures found at operation may be ascribed to a faulty interpretation of these cicatricial tissue changes. If not operated on, the paralysis ultimately disappears.

It has long been recognized that these birth cases have a pathology similar to that of many adult cases following injury to the shoulder region. The pathology of the adult cases has never been established and up to the present time is being variously interpreted. Erb established his theory of a rupture at the junction of the fifth and sixth cervical roots of the brachial plexus on his four adult cases. His single obstetrical case was incidental. Yet his theory has lived only in connection with the obstetrical cases. In connection with adult cases it has long been obsolete.

The Duchenne-Erb theory of a rupture of the brachial plexus so localized as to produce a paralysis of the same four or five brachial muscles every time has been widely accepted but not corroborated. The only exact scientific evidence on which this theory is resting is the finding of this exactly localized paralysis by electrical examination in four cases of obstetrical paralysis by Duchenne and in one by Erb, and in Erb's four adult cases.

The support of this theory obtained by experimental work on infant cadavers is open to some question. Most, if not all, experimenters have admitted that "great force must be employed to produce the lesion." The author tried direct extension on the head and lateral bending of the neck on both sides of the neck in six infant cadavers, with probably much more force than any competent physician has ever employed in a successful delivery, without obtaining the slightest evidence of a rupture of the brachial plexus.

Thomas, Philadelphia.

KYSTE CONGENITAL INTRASPINAL PROBABLEMENT MENINGOCELE ANTERIEURE FERMEE (CONGENITAL INTRASPINAL CYST PROBABLY A MENINGOCELE CLOSED ANTERIORLY).

Knud H. Krabbe, Rev. neurol. **26**:561 (July) 1919.

The author reports a case of congenital cyst of the spinal cord with symptoms appearing shortly after birth. The principal points in the case are: Fundus, normal; fontanel, normal; neck, short; kyphosis at the dorsal curvature; abdomen, large and flabby, showing paralysis of the abdominal muscles; lower extremities and trunk analgesic up to nipple line. Above this line there was normal reaction to pin prick. Roentgen-ray examination of the spinal column was negative. There was paraplegia but no atrophy. One month after birth there were hypertonia and contracture of the lower extremities. There were no fibrillary twitchings. The muscles of the face and upper extremities showed normal reactions to galvanic and faradic currents. Function of the sphincters was normal, but the bladder was occasionally distended. There was no decubitus and no vasomotor disturbance. The spinal fluid on two occasions was tinged with blood and later feebly tinted yellow. Culture of the fluid was negative. Intellectual, or rather mental, reactions were apparently normal. There were progressive but gradual weakness, loss of weight, cyanosis, and finally death. Laboratory examinations were negative, except for a slight eosinophilia.

Necropsy examination revealed bilateral bronchial pneumonia; abdominal organs negative; brain, microscopically negative. At about the fifth dorsal segment, in the leptomeninges, anteriorly, there was a cyst 2 cm. long, 6 mm. broad and 4 mm. thick. This cyst exerted pressure on the cord which, in section at the level of the cyst, took the shape of a crescent moon. Below the cyst there were adhesions between the leptomeninges and the dura mater. Microscopic examination showed no evidence of tumor or hemorrhage. In the cervical and upper dorsal regions of the cord there was marked degeneration in the columns of Goll, more marked toward the external portion of the cord. There was an increase of neuroglia in the degenerated areas. The myelin sheaths in the lateral cerebellar columns and in the tract of Gowers was well preserved, as well as in the anterior columns; elsewhere, they were in great part destroyed. Much of the gray matter and of the posterior columns showed neuroglia proliferation. The central canal was flattened but open. Below the cyst there was a noticeable degeneration of the pyramidal tracts with increase of neuroglia; this was also true of the anterior columns, and of the anterior horns. The sacral spinal cord showed a number of small defects completely filled with compact connective tissue and the motor cells of the anterior horns completely destroyed.

The author's conclusion is that the case presents a cystic formation, congenital, in the sense that the symptoms appeared so rapidly after birth. He rules out hemorrhage and necrotic tumor. He states that he has not succeeded in finding in the literature any description of a congenital cyst in the anterior portion of the cord. He grants that it might be considered a variety of meningocele. Surgical intervention might have resulted in improvement, but laminectomy in so young a child would have been dangerous. Difficulties in diagnosis lie between hematomyelia, cyst and hematomyelia combined with tumor. The history led the author to diagnose the condition as hematomyelia. What necropsy examination showed has already been described, and constitutes the reason for reporting the case.

Jones, Detroit.

PARAPLEGIA AFTER ARSPHENAMIN. T. J. Dimitry, J. A. M. A. **74**:1150 (April 24) 1920.

Dimitry reports the case of a man who suffered a complete loss of sight before other symptoms developed to make possible a diagnosis, though some precursory symptoms obtained in his history were not recognized at the time as being important. Fundus examination elicited a distinct elevation of the optic nerve of both eyes. This elevation was greater in the right eye. The pupils were widely dilated and did not respond to light. Tests for syphilis were negative. Following the intravenous injection of 0.06 gm. of arsphenamin, the patient complained of stiffness in the legs and pain in the back. He could not move his right leg, and during the afternoon this paralysis extended to the other leg. It was necessary to catheterize the bladder and later to contend with an overflow of the urine. He suffered an inability to evacuate the bowels and subsequently an incontinence of feces. He developed decubitis and a painful hyperesthetic area of the left arm. The paralysis of the legs was absolute, and all voluntary motion and sensation in all its qualities, including temperature and touch, was destroyed. Different degrees of deep and superficial anesthesia and hyperesthesia extended to within an inch of the nipple anteriorly and not quite so high posteriorly. The reflexes were abolished in the legs with a flaccid paralysis and the appearance of a rapid wasting. Two days after the injection of arsphenamin, the elevation in the optic nerve had improved and within the next day had disappeared entirely, leaving a normal appearing optic nerve, though the patient was blind. In spite of the apparently harmful effect of the arsphenamin, a second dose of 0.06 gm. was given and a dose of 0.09 gm. of neo-arsphenamin was given a few days later. Mercury was given by inunction and the iodids were used in increasing doses. For weeks the patient took 75 drops of saturated solution of potassium iodid three times a day. Seven months later the patient could walk and had practically perfect vision. There remained some bladder disturbance. The Wassermann reaction with 0.75 c.c. of spinal fluid was negative. The cell count was 173; of these cells, 95 per cent. were lymphocytes. The colloidal gold curve was similar to the curve found in tuberculosis. The blood Wassermann reaction was negative.

The author is of the opinion that arsphenamin played no part in the development of the myelitis. He regarded the case as one of retro-bulbar optic neuritis with myelitis, which is a definite clinical entity. A brief review of the literature of this condition is made and also a review of cases of paralysis following the use of arsphenamin.

Klauder, Philadelphia.

RECHERCHES EXPERIMENTALES SUR LE VIRUS DE L'ENCEPHALITE LETHARGIQUE (EXPERIMENTAL RESEARCH REGARDING THE VIRUS OF EPIDEMIC ENCEPHALITIS). C. Levaditi and P. Harvier, Bull. de l'Acad. de méd. **83**:365 (April 20) 1920.

In February, 1920, these workers succeeded for the first time in inoculating a rabbit with virus from a patient with epidemic encephalitis. The material used consisted of an emulsion of the cerebral gray substance (cortical, mesencephalitic and bulbar) obtained at necropsy from a woman of 46 who had died of encephalitis on the ninth day. In this patient there had been fever, facial herpes, light somnolence, aphasia, carphologic movements, urinary retention and slight lymphocytosis of the spinal fluid. (Necropsy confirmed the clinical diagnosis and showed inflammatory and perivascular alterations located chiefly in the bulb and Broca's convolution.)

Two tenths of a cubic centimeter of the emulsion was inoculated into the brains of each of two rabbits and a monkey. One of the rabbits died on the eighth day. Cultures of the brain and blood remained sterile. Histologic examination showed characteristic meningeal encephalitis lesions of the cortex and mesencephalon. The other two animals survived without symptoms.

An emulsion of the nerve centers of the rabbit which had succumbed, was inoculated, in the dosage first used, into the brains of two rabbits. These died on the sixth and seventh day, showing the identical lesions.

The following findings and conclusions are summarized:

1. The period of incubation of the sickness after intracerebral inoculation is four or five days. In animals, the morbid symptoms, torpor and epileptic, myoclonic or choreic movements, appear only a few days before death.
2. The virus survives in glycerin.
3. The virus is filtrable (Chamberland 1 and 3).
4. In the rabbit, the virus is inoculable by way of the peripheral nerve (sciatic) and by way of the anterior chamber of the eye.
5. The virus does not appear pathogenic for the monkey when obtained directly from man but becomes so after it has been subjected to a certain number of passages through rabbits.
6. After these passages, the virus becomes pathogenic for the guinea-pig.
7. The virus preserves virulence after desiccation.
8. The virus exists in the spinal cord of animals inoculated by the cerebral route.
9. The serum of patients recently convalescent (for a month) from epidemic encephalitis possesses no neutralizing action on the virus.

Davis, New York.

NEVRITE HYPERTROPHIQUE PROGRESSIVE NON-FAMILIAL DE L'ADULTE (NONFAMILIAL PROGRESSIVE HYPERTROPHIC NEURITIS OF ADULTS). G. Roussy and Lucien Cornil, Ann. de méd. **4**:296, 1919.

These authors report a case which differs in certain respects from the two known types of the disease. The first type, that of Déjerine and Sottas, is characterized by general muscular atrophy with fibrillary twitching, hypertrophy of nerve trunks, shooting pains, ataxia, Romberg's sign, nystagmus, myosis and Argyll Robertson pupils. The pathologic basis for this is a hypertrophic interstitial neuritis. The second type, that of Marie and Boveri, is characterized by muscular atrophy, more marked in the lower extremities than in the upper, kyphoscoliosis, exophthalmos, intention tremor and jerky speech recalling that of multiple sclerosis. The pathologic basis is an involvement of both nerve fibers and interstitial tissue.

A summary of the case follows: The onset was at 40 years of age. The man had no relations similarly afflicted. There was atrophy of the Aran-Duchenne type in the upper extremities with fibrillary twitching and reaction of regeneration. Ataxia was present in all extremities. Reflexes: Plantar and Achilles' reflexes were absent; the right knee jerk was diminished, the left, absent. The pupils were normal. Pathology: (1) degeneration of the myelin sheaths with onion shaped enlargement at intervals; (2) increase of the number of the nuclei of the sheaths of Schwann; (3) alterations of the axis-cylinders; (4) presence of many young regenerating axis-cylinders; (5) mild hyperplasia of the interstitial tissue; little or no vascular change.

Compared to the Déjerine-Sottas type, this case showed no generalized muscular atrophy, no myosis and no Argyll Robertson pupils or nystagmus, while intention tremors were present. It began later in life.

Compared to the Marie-Boveri, type there was no familial history, no scanning speech or exophthalmos, while an Aran-Duchenne type of atrophy was present.

The authors suggest the title of this paper as a name for this disease.

Kraus, New York.

SUR LES TROUBLES DE LA CORRELATION CARDIO-RESPIRATIORE DANS LA NEUROPATHOLOGIE (DISORDERS OF CARDIO-RESPIRATORY CORRELATION IN NEUROPATHOLOGY). J. Parhon, L'Encephale **15**:185 (March 10) 1920.

One knows that the normal functioning of respiration determines, during inspiration, the virtual formation of an intrathoracic vacuum and that in this manner the emptying of the blood from the head and general venous system is facilitated. Each time that the inspirations are not sufficiently complete, in addition to a sensation of oppression or need of air, a passive venous congestion of the head and brain results, with augmentation of intracranial tension. The writer expresses his belief that this congestion and augmentation of intracranial tension seems to contribute to the explanation of certain pathologic phenomena of the nervous system. Among those phenomena are the well-known psychic troubles which one sees in cardiac cases and also such special phenomena as fascicular tremors, neurasthenic feelings of fulness in the head and certain other abnormal sensations. He points out that neurasthenics, in general, often present an insufficiency in amplitude of the respiratory movements. He has seen fascicular tremors, approaching clonic convulsions, lasting several minutes and localized in a single muscle. Abnormal sensations, especially sensations of localized engorgements, are considered phenomena in the domain of sensibility of the same order as the fascicular tremors in the domain of motility. Is there an excitation of cortical centers by the congestion — perhaps even microscopic hemorrhages — and also an acidification of the blood?

A disorder in the cardiorespiratory correlation also appears to exist in migraine. In one case, as the respirations became ample and regular, the headache diminished and then disappeared.

The sign of Bryson (the diminution of the amplitude of respiratory movements) in Basedowians is accompanied by anxiety, and this accords with the writer's conception. He says, "I have, for a long time, held that modifications of respiration have an important part in the mechanism of affective phenomena."

Davis, New York.

LA FORME DE L'IRREGULARITE PUPILLAIRE SYPHILITIQUE (THE FORM OF THE IRREGULAR SYPHILITIC PUPIL).

E. Rasquin, Ann. d'Oculistique **157**:162, 1920.

The conclusions drawn are based on 824 cases of irregular pupils of clearly demonstrable syphilitic origin. Particular attention was paid to the form of the irregularity, eliminating of course cases due to synechia even in the presence of syphilis.

In 107 cases the form described by Brown-Séquard and Terson as being an early and characteristic symptom of tabes was found. This consisted most frequently in the elliptical or oblong type. It is recognizable in daylight as well as in the dark room and usually is present in both eyes.

In his experience this irregularity is not a morbid entity, except when pushed to an advanced degree; in fact, normal pupils are not always perfectly round, but may be slightly oblong usually in the vertical direction.

When the irregularity is pronounced there is usually an alteration in the light reflex.

Seventy-nine per cent. of cases examined showed a form with large curved angles. At times the deformity extended almost completely around the pupillary circle, giving the appearance of a polygon with curved sides. This type exists in all forms of syphilis, even before any other pupillary symptom. It is usually bilateral, but may be unilateral.

Then there are the undefined types, such as notches and sharp angles. As a rule, these forms accompany anisocoria and alteration of the pupillary reflex, in which the syphilitic process is far advanced.

Importance is attributed to the irregular pupils because: (1) It is a condition present in all stages of syphilis. 2. It is present in 79 per cent. of cases of true syphilis. 3. It represents the minimum amount of waste in the search of irregular pupils of syphilitic origin. The author personally considers this form of irregularity, especially when bilateral, as a quasi certain symptom of syphilis. Two cases, from among many observed, are reported to substantiate his belief.

Scarlett, Philadelphia.

LA RESTAURATION FONCTIONNELLE DANS LES COMMOTIONS MEDULLAIRES (FUNCTIONAL RESTORATION IN SPINAL CONCUSSIONS).

André Barbé, Bull. méd. **34**:373 (April 21) 1920.

The author was permitted to follow step by step the functional restoration in a case showing spinal concussion.

In the middle of June, 1918, S. was wounded in the middle lumbar region by a bullet. At operation this was found at the level of the inferior border of the eleventh dorsal vertebra, having fractured the twelfth. That the cord was not actually attained was shown by the fact that it was possible to extract the bullet without at any time opening the spinal canal. No spinal fluid escaped.

The immediate symptoms from the injury included paraplegia, abolition of knee and plantar reflexes, sensory losses over the lower extremities and retention of urine and feces. Seventeen days after the wound was received the retention was succeeded by incontinence. The sensory loss comprised absolute anesthesia over the legs up to the region of Poupart's ligament. Sixty-five days following the receipt of the injury the patient could make some flexion movements of the thigh on the pelvis; the sensory condition was the same; both knee reflexes were absent; the left ankle reflex was weak, the right normally active. There was no clonus but the Babinski sign was positive on each side. Five months and a half after the injury all movements of the thighs had returned. The sensibility was then characterized by a hypesthesia of the lower half of each leg, accompanied by a thermo-anesthesia in the same area, and by loss of deep sensation. Both the knee and ankle reflexes were absent. The Babinski reflex was positive on the right and negative on the left. The sphincteric disturbance had disappeared. Seven months after his wound, the patient could walk with two canes and while lying down he could execute all movements in both feet. The improvement in the extensors was clear. Deep and superficial sensibility had returned. The knee reflex was now obtained on the right. The Babinski reflex was positive only on the right. Atrophy was greater on the left.

The writer points out the parallelism between the motor and sensory recovery. In each, the return of function progressed from the proximal portion of the leg to the peripheral. Further, superficial sensation improved more promptly than the deep.

Davis, New York.

RECHERCHES SUR LA PATHOGENIE DE LA CRISE DE MIGRAINE (INVESTIGATIONS INTO THE PATHOGENESIS OF THE ATTACKS IN MIGRAINE). P. Pagniez and A. Nast, Presse méd. **28**:253 (April 28) 1920.

Anaphylactic phenomena in epilepsy have been previously reported by Pagniez (*Presse méd.* **27**:693 [Nov. 19] 1919, abstracted in the Archives **3**: 328 [March] 1920), and a case of migraine is discussed in connection with similar findings. The subject of the experiments had had typical attacks of migraine for years, and had found that the ingestion of chocolate, especially with milk, would precipitate an attack, but that a small dose (0.5 gm.) of peptone taken before each meal would prevent attacks even though chocolate were eaten.

The experiments consisted in giving chocolate and milk, with or without other foods, before and after discontinuing the preventive medication. Differential leukocytic counts were made after meals to demonstrate any anaphylactic evidence ("crise hémoclasique initiale" of Widal) in the blood picture. Normal digestive leukocytosis and no migraine were found following meals that included milk and chocolate, both when the preventive medication was being administered, and for several days after its withdrawal. Nine days after, however, the experiment showed instead a leukopenia (to 3,700), followed several hours later by typically severe migrainous symptoms.

The results illustrate a temporary actual immunity sometimes conferred by a drug, the existence of a period between attacks when a subject is not susceptible to a known exciting cause, and finally the period of "potential migraine," when an attack will not arise spontaneously but will follow the action of a known excitant.

Huddleson, New York.

CONTAGIOSITE DE L'ENCEPHALITE LETHARGIQUE (CONTAGIOUSNESS OF EPIDEMIC ENCEPHALITIS). Arnold Netter, Bull. de l'Acad de méd. Par. **83**:*373* (April 27) 1920.

The author considers that a case of epidemic encephalitis which he observed was probably the first case in Paris; therefore there is special reason to believe that two other cases which developed in the same household developed with that case as the initial focus of contagion. He relates, from the experience of others, seven instances in which contagion seemed equally clear. In contrast, however, he records that though twenty-one cases of epidemic encephalitis in children were, because of lack of space, placed in general wards in the hospital, no spread of the disease inside the hospital occurred in any instance; also, among 107 cases in private families, there was only one instance of two patients in a single family. Further, he quotes figures published in England. Among 368 cases, five times only did more than one case occur in one home.

He says the contagiousness of epidemic encephalitis is difficult to prove as is that of cerebrospinal meningitis and of poliomyelitis.

He insists on the presence of the virus in the salivary glands in encephalitis. His collaborator, Henri Durand, has studied a parotid gland of a fatal case and has found in the interlobular spaces, around the vessels, excretory ducts and acini, numerous cellular masses (mononuclears). The lesions resembled those found in the same location in hydrophobia by Manouëlian. Once this localization of the virus is accepted, the saliva becomes one factor of contagion.

The author concludes:

Epidemic encephalitis is contagious. It is carried by the saliva.

By reason of the long persistence of the virus in the nerve centers the patient is for a long time capable of transmitting the affection. There are reasons for thinking it can be transmitted by a convalescent, and also by an aborted case or by a healthy person who has become a carrier. It is, therefore, necessary to search out all cases, both established and suspected. However, in our present state of knowledge one would not advise isolation when in the absence of all precaution, the great majority of cases occur singly.

Davis, New York.

NERVE DEAFNESS DUE TO CONGENITAL SYPHILIS IN THREE CHILDREN. M. B. Kay, J. A. M. A. **74**:1162 (April 24) 1920.

The cases reported by Kay are remarkable not because syphilis produced deafness, but because it attacked all three children of one family in the same manner, every other part of the body escaping but the eighth nerve. The ages of the children were 7, 5 and 4, respectively. The onset of deafness was between the age of 1 and 2. The blood Wassermann test of the elder child was ++++. No mention is made of the result of this test in the other two. The father admitted having had a genital lesion. The blood Wassermann test of the parents were ++++. Owing to the fact that at least a year had elapsed before signs of deafness developed, Kay believes that these are undoubtedly cases of "late hereditary syphilis." Each of the children was given ascending doses of potassium iodid, up to 35 grains, three times a day; one-half grain of mercury with chalk, three times daily, was also administered. The general result was good.

Klauder, Philadelphia.

L'HYPERGLYCORACHIE DANS L'ENCEPHALITE EPIDEMIQUE (HYPERGLYCORACHIA IN EPIDEMIC ENCEPHALITIS). C. DOPTER, Bull. de l'Acad. de méd., Par. **83**:203 (March 2) 1920.

In agreement with other investigators, the writer has often found the sugar content of the spinal fluid increased in epidemic encephalitis. In a case which he reviews the reading was 95 cg. glucose per liter. Separation of epidemic encephalitis from various types of meningitis is at times difficult. Tuberculous meningitis, which is the type most apt clinically to simulate epidemic encephalitis, shows either diminution or absence of sugar from the spinal fluid. In syphilitic meningitis the sugar remains normal (about 50 cg. per liter).

The writer recalls that hyperglycorachia is present in diabetes, sometimes in uremia, in pneumonia and bronchopneumonia, in Malta fever, in hydrophobia, in whooping cough, in some cerebral tumors, in some hemorrhages (cerebral), in amyotrophic lateral sclerosis and in some cases of chronic cerebrospinal syphilis. Therefore it is not pathognomonic for epidemic encephalitis It is only from the point of view of differentiation of this affection from meningitis that it remains of interest.

It is found hyperglycorachia is not a constant finding in all cases. Without data on which to base an answer, the author raises the question whether it is only in cases with bulbar involvement that the sugar increase occurs.

DAVIS, New York.

SUR LA COMPOSITION CLINIQUE DU LIQUIDE CEPHALO-RACHIDIEN DANS UN CAS D'ENCEPHALITE LETHARGIQUE (THE CHEMICAL COMPOSITION OF THE SPINAL FLUID IN LETHARGIC ENCEPHALITIS). P. MARIE and MESTREZAT, Bull. l'Acad. de méd. **83**:103 (Feb. 3) 1920.

The following table is given, showing the results of chemical analysis of spinal fluids:

	Spinal Fluid Found with Lethargic Encephalitis	Spinal Fluid of Normal Persons
Albumin	0.14	0.15-0.30
Fibrinogen	0.0	0.0
Chlorids	7.25	7.30
Dry Extract	10.45	16.90
Mineral matter	8.75	8.75
Urea	0.326	0.20
Acetone	*	....
Sugar	0.94	0.53

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* Very small quantity.

Other data are given to show that in lethargic encephalitis the albumin and chlorids of the spinal fluid are normal and the sugar increased while in tuberculosis and the acute meningitides, the albumin is greatly augmented, the chlorid much diminished and also the sugar greatly decreased or even absent.

P. Marie presents these figures to emphasize that lethargic encephalitis lacks the signs of meningitis. He thinks that the Kernig sign and rigidity of the neck are absent. Regarding the cell count in the spinal fluid he reports a moderate increase with 26 the highest count encountered.

DAVIS, New York.

ONE THOUSAND PSYCHIATRIC CASES FROM THE CANADIAN ARMY. C. K. Clark and C. B. Farrar, Canad. J. Ment. Hyg. **1**:313 (Jan.) 1920.

The authors add more facts to the evidence in favor of certain essential principles of psychiatry:

1. Satisfactory work depends, among other things, on reception wards, hospitals or beds.
2. Stress (of war) brings out in most people diseases whose causes were present in times antedating the stress.
3. Malingering is prima facie evidence of mental abnormality.

The local situation is discussed in some detail, showing how "shell shock" cases have been kept out of the older hospitals for mental diseases to which most patients with dementia praecox have gravitated.

Bond, Philadelphia.

SOME NOTES ON ASEXUALIZATION; WITH A REPORT OF EIGHTEEN CASES. Martin W. Barr, J. Nerv. & Ment. Dis. **51**:231 (March) 1920.

Barr makes a plea for the surgical asexualization of mental defectives. His strongest argument is the alarming prevalence and probable increase of this class, there being between 300,000 and 400,000 mental defectives in the United States. It is estimated that less than 40,000 of this number are being cared for in institutions.

Eighteen cases are cited to show the beneficial results of asexualization. In addition to the removal of all danger of procreation, there was also a general intellectual and behavior improvement. It is interesting to note that the legislatures of thirteen states have legalized asexualization of imbeciles, criminals and rapists.

Strecker, Philadelphia.

LE TRAITEMENT DE L'ENCEPHALITE LETHARGIQUE (THE TREATMENT OF EPIDEMIC ENCEPHALITIS). M. Arnold Netter, Bull. de l'Acad. de méd. **83**:303 (March 30) 1920.

The writer recommends the intraspinal injection of serum of cured cases in the treatment of epidemic encephalitis. Also, he recommends the administration of hexamethylenetetramine by mouth. Discussing drug therapy with arsenic and antimony, he reports one unfavorable result from the intravenous use of arsphenamin and intends to report later regarding the effects from tartar emetic.

In accordance with a belief that the virus of hydrophobia and poliomyelitis are often present in the buccal cavity, especially in the parotid and submaxillary glands, he suggests that the virus of epidemic encephalitis, a similar disease in a sense, may also be harbored there. He suggests that increasing salivary flow may hasten the elimination of the virus and therefore recommends the administration of jaborandi.

The author obviously places most value on the artificial induction of abscesses. For this purpose he injects 1 to 2 c.c. of essence of terebinthina into a buttock. He believes this procedure saved lives in the grip epidemic and that "the effect is not less marvelous in epidemic encephalitis."

Davis, New York.

LES HALLUCINATIONS LILLIPUTIENNES (LILLIPUTIAN HALLUCINATIONS). Leroy and Rogues De Fursac, L'Encephale **15**:189 (March 10) 1920.

Lilliputian hallucinations comprise a variety of visual hallucinations of a particular character and, as the name implies, are small personages of a finger's height accompanied sometimes by small equally diminutive animals in the proper proportions. The patient believes that he has before his eyes a world created by Swift in Gulliver's Travels. These small personages are multiple, moving, fugitive and usually dressed in many colors. They form a veritable theater of marionettes—not flat as the figures on the screen, but in true perspective and relief. Often appearing alone, they are sometimes preceded or followed by visual hallucinations of a different, more usual kind. Though these hallucinatory midgets seem to the patient to be only a few centimeters high, he sees them capering over furniture the correct size of which is normally appreciated. In this it is not a diffuse "micropsy."

These hallucinations are usually accompanied by an agreeable affective state. They obviously amuse the person who sees them. They occur in toxic states (alcohol, chloral, ether) or in toxic infectious conditions (cholera, typhoid fever). The author agrees with Leroy, who first pointed out the toxic pathogeny of the syndrome.

Davis, New York.

Society Transactions

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, March 26, 1920

S. D. Ingham, M.D., *President*

A CASE OF TUMOR OF THE LUMBAR ENLARGEMENT OF THE SPINAL CORD. Presented by Dr. J. Hendrie Lloyd.

H. Williams, B., a man, aged 43, had a negative history except for a chancre nineteen years before. His trouble began with a burning sensation in the toes of the right foot with foot drop, then numbness in the limb and motor inability ascending to the hip. There were numerous fibrillary tremors in the thigh, then pain in the sacral region and in a few weeks dysuria. Hyperesthesia of both legs then appeared; even the bed clothing distressed him. The hyperesthesia in time disappeared and was replaced by anesthesia. Complete paralysis of the bladder and bowel supervened. Six months after the onset of the disease, he was paraplegic with total paralysis of bladder and bowel. There was marked decrease in tone of the paralyzed muscles, especially of the right leg, and atrophy and active fibrillations in the affected muscles. The cremasteric reflexes were preserved; all the deep reflexes abolished. There was total anesthesia of the right lower limb behind up to the crest of the ilium, but in front only to a little above the knee; on the left side tactile anesthesia extended only a little above the ankle. There was a rather extended area of hyperesthesia above the anesthesia, and the patient complained much of pain in this area, especially in the right leg. The cranial nerves were normal. The Wassermann test of the blood and spinal fluid was negative. Roentgen-ray examination of the lumbar spine and sacral region was negative.

Evidently there was a lesion of either the lumbar enlargement or cauda equina. The onset with pain, the gradually progressive course and the rather marked unilateral character, indicated that it was a neoplasm rather than a meningomyelitis. Especially noteworthy, however, was the active fibrillation of the muscles of the thighs, such as is seen in chronic anterior poliomyelitis and which must be rare in tumor of the cord. The distribution of the anesthesia also indicated a lesion located to the right. It involved the sacral nerves and the lumbar as high as the second or third segment. The atrophy, flaccidity and abolished deep reflexes also pointed to a lesion of the lumbar cord.

The patient died a few days after admission to the Philadelphia Hospital. Necropsy revealed a large growth embedded in the lumbar enlargement; it was not a meningeal growth, but a true medullary one. The membranes were not extensively involved and the bones of the spine not at all. The appearance of the growth was that of a gumma. There were other important findings: The chain of retroperitoneal lymph glands in the lumbar region, extending down into the pelvis, was greatly enlarged, forming a huge mass.

There was an old tuberculous lesion of the apex of the right lung, and in the right lobe of the cerebellum was found a growth as large as a marble and of similar appearance to the one in the lumbar cord.

Dr. B. L. Crawford sent me this statement: "Sections of enlarged post-peritoneal lymph nodes show marked fibrosis with areas of caseation and intense mononuclear cell infiltration. The cells are more numerous at margin of nodes, and in places form small foci containing giant cells.

"The cross section of enlargement in the cauda equina shows a diffuse fibrosis and necrosis, with intense mononuclear cell infiltration at outer margin of cord and in the meninges, forming numerous foci in which giant cells may be seen. No nerve fibers remain.

"Section of nodule at base of brain shows a similar chronic inflammatory process, fibrosis, necrosis, mononuclear cell infiltration, giant cells and localized meningitis.

"Diagnosis: Chronic fibroid and caseous lymphadenitis, tuberculous, post-peritoneal. Tuberculoma in cauda equina and in cerebellum, under the surface of the right lobe with localized meningitis."

Dr. Crawford said: "I have termed the lesion in the cord tuberculous. I realize the strong possibility that it may be syphilitic, but in view of the widely disseminated tuberculosis elsewhere, I consider the diagnosis the more logical. I have been unable to demonstrate tubercle bacilli in the section." He said that he had not had an opportunity to stain the spirochetes.

Was the cord lesion tuberculous or syphilitic? Pathologists seem to agree that it is not always easy to distinguish these two conditions. The intense lymphocyte infiltration is much like that which is seen in gummas — most marked in the membranes and outer portions of the growth and in the walls of the vessels. Giant cells are also not unknown in gummas.

Solitary tuberculoma of the spinal cord has been observed. In a series of fifty cases of cord tumor, tabulated by Dr. Mills and the writer in Pepper's "System," four such cases (or 8 per cent.) are noted. Nevertheless, in the present case I favor gumma. The coexistence of syphilis and tuberculosis in the same patient has been observed, and in a recent paper Dr. E. H. Funk of this city (*Penn. M J.*, March, 1920, p. 310) says that such coexistence is not uncommon. The presence of a large mass of caseating tuberculous lymph glands in the retroperitoneal space is therefore not altogether conclusive evidence that the cord tumor is not a gumma.

DISCUSSION

Dr. S. Leopold said there was no way of diagnosing this specimen except by staining for the organism. He said he noticed in the lymph node the daughter tubercles which he did not think were found in a gumma. He thought, therefore, that this was a tuberculoma rather than a gumma.

Dr. W. G. Spiller said he was much more inclined to think that the growth was a tubercle. Tubercles were often multiple, occurred in different parts of the nervous system and were not uncommon in the cord. A distinct gumma of the cord was of great rarity. Dr. Spiller said he had not in his collection of over 800 specimens a single gumma of the spinal cord; he was not referring to gummatous meningitis.

Dr. J. Hendrie Lloyd said that he did not understand how any one could be dogmatic about the tissue under the microscope. It was an amorphous, breaking-down tissue, undergoing fibrosis, and around the edges there was intense small cell infiltration, which was characteristic of gumma. Dr. Lloyd found that the greatest reliance was placed by pathologists on the presence of giant cells. There, he confessed, the appearance would indicate that it was tuberculous, although giant cells are also seen in gumma.

THE CLINICAL INVOLVEMENT OF THE PERIPHERAL NERVES IN DIABETES MELLITUS. Dr. Walter M. Kraus.

Dr. Kraus presented the results of a statistical survey of 450 case reports of diabetes mellitus. They were selected from nearly 700 accumulated during ten years in the Metabolic Department of the Vanderbilt Clinic, New York.

The discussion was limited to those cases having symptoms pointing to disease of the spinal cord or peripheral nerves. The symptoms and signs considered were: areflexia, neuralgia, ulceration and gangrene, pseudotabes, spinal cord disease, herpes zoster and peripheral neuritis.

Absence or diminution of knee jerk on one or both sides was found in 30 per cent. of the cases; exaggeration 10 per cent. The Achilles reflexes were absent in 64 per cent. of the cases tested (8 per cent.).

Age, sex, race, nutrition (determined by loss of weight and the weight-height index), duration of the disease, condition of the arteries and the blood pressure appeared to play no part in determining hypoflexia or areflexia. The severity of the disease, as determined by the course and carbohydrate tolerance, seemed to play a part since knee reflexes were absent in 22 per cent. of the mild cases, 33 per cent. of the moderately severe cases and 50 per cent. of the severe cases. Neuralgic pains were present in the extremities in about 25 per cent. of the cases.

Cases showing changes in light touch sensation and reflexes with some neuralgia, were not uncommon. Probably these symptoms were due to spinal cord involvement. This had been shown pathologically by Williamson, Kalmus, Schweigger and others. These cases were not "neuritis." Peripheral neuritis in diabetes is rare. None of the pathologically reported cases were true sensorimotor peripheral neuritis. In the 700 cases at the Vanderbilt Clinic there was only one. The patient was also alcoholic. The coincidence of alcohol in the few cases showing peripheral neuritis is emphasized by all writers on the subject.

Ulceration and gangrene occurred in few cases. Herpes zoster in none.

The cases showing absence or diminution of deep reflexes, sensory changes in the skin, neuralgia and ataxia (diabetic pseudotabes) can all be explained anatomically by a partial lesion of the intramedullary portion of the posterior roots. The changes in the posterior columns follow. Gangrene may have the same origin.

DISCUSSION

Dr. W. B. Cadwalader said that if an endogenous toxic process is assumed to be going on in the posterior columns of the cord in cases of diabetes resembling that found in pernicious anemia, the loss of the tendon reflexes should not be expected until after the sensory symptoms become firmly established. In pernicious anemia the tracts situated close to the midline in the posterior columns are affected early and as the process extends laterally the fibers of the reflex arc are destroyed late and the patellar tendon reflexes, which are generally exaggerated at first, become diminished or lost entirely. In Dr. Kraus' cases of diabetes the tendon reflexes were lost early and the mild sensory changes developed later, so that this order of development of symptoms would suggest a morbid process beginning in the region of the posterior root entrance zone of the spinal cord and extending toward the midline—exactly the reverse to the order in which the signs of pernicious anemia develop. It would be interesting to know whether in certain cases patients with diabetes, when profoundly toxic and without tendon reflexes, recovered their tendon reflexes when toxemia disappeared.

Dr. S. Leopold said there was one diagnostic point that might be considered. As none of Dr. Kraus' patients had come to necropsy, he would like to ask whether he had performed lumbar puncture on any of them. In the exogenous changes one usually finds a round cell infiltration and an increased number of cells in the spinal fluid. If endogenous, the spinal fluid should be negative.

Dr. Kraus said that Williams of Manchester, England, who has done the most work on the subject, believed that the posterior roots were affected exclusively in their intramedullary course.

The patients in question recognized the position of their fingers and toes quite well.

The pains were thought to be due in some cases to arteriosclerosis. The only positive finding obtained from the records was that the knee-jerks were absent twice as often in severe cases as in mild cases.

TUMOR OF THE CRANIUM IN THE PARIETAL REGION. Presented by Dr. John H. W. Rhein.

S. M., colored, aged 49, with negative family and previous history, seven years ago was struck by a club over the left parietal region. Two years later a small tumor appeared at the site of the injury, which gradually grew larger, more rapidly in the last two years. Two years ago, he had a convulsion, probably general. He was confused for two hours after the attack, his speech was thick, his right arm and leg were powerless. He complained of numbness in the right hand for a week or two and dragged his right foot. This was followed by several attacks in which the right side of the face would twitch, there was some disturbance of speech, and he could not express himself clearly. The attacks were preceded by pain in the right parietal region at the site of the tumor. These minor attacks occurred about twice a month.

In the autumn of 1918, he had a second convulsion. The right arm and leg have remained weak ever since. He had a third convulsion in the summer of 1919 and a fourth in October. During these attacks he twitched on the right side only. Since the attack in the summer of 1919, he has had difficulty in recalling words, dates, etc. He becomes confused when going out into the street and is not able to find his way home.

When he presented himself on November 11, he complained of not seeing well, difficulty in recalling words and pain in the tumor over the left parietal region. Examination revealed: The tongue was protruded in median line; both sides of the upper and lower face moved equally well and the masseters were normal. The right pupil was slightly larger; both responded promptly to light and in accommodation. There were no sensory disturbances in the face. The dynamometer registered 100 on the right, 115 on the left. The arm reflexes were prompt and equal, knee reflexes were increased more on the right than on the left. The right ankle reflex was more active than the left. Station was good. In walking, the right leg was suggestive of the hemiplegic gait and was distinctly weaker than the left. There were no atrophies. Astereognosis was present in the right hand. He felt the pin prick equally well on both sides, but examination of the sensation was not entirely satisfactory owing to a somewhat disturbed mental condition. The Wassermann reaction was moderately positive. The eyegrounds were negative. Hypermetry was present on the right side. Roentgen-ray examination revealed an extensive lesion in the right parietal region covering an area of 6 inches in length by 4 inches in width, showing overgrowth of bone and a destructive process. The tumor was immobile, somewhat soft in places but not fluctuating. The integument could be moved fairly well over the tumor. It was not sensitive and did not pulsate. The tumor measured 13 cm. in its anteroposterior diameter and 10 cm. in its transverse diameter.

On Jan. 3, 1920, the patient had an attack in which he lost consciousness, followed by paralysis of the right arm and leg and inability to talk, which lasted for two days. In a week he was able to walk, dragging his foot; the right arm was weak.

On January 18, Dr. Appleman found a right homonymous hemianopsia. The eyegrounds were normal.

On February 24, a short time after a convulsive attack, it was noted that the lower part of the face on the right side was paralyzed. He dragged his foot; the grasp on the right side was weaker, and he exhibited marked apraxic phenomena. In attempting to comb his hair, he made stroking movements 3 or 4 inches away from his head. When asked to brush his clothes with a clothes brush, he made purposeless movements in the air. A key was placed in his hand and he was asked to unlock a door. Instead of the proper movements, he placed the key on his nose or made purposeless, irregular movements, pointed at the door and suddenly, after a long effort, plunged the key spasmodically into the keyhole, after balancing it before the keyhole and assisting with the left hand. The Wernicke pupillary inaction was not present. He could not answer questions intelligently, apparently because he could not recall the proper words. He was able to read letters but was agraphic.

Examination on March 26 showed that the right arm was distinctly weaker than the left. There was practically no evidence of apraxia. The reflexes were increased everywhere, and there was a tendency to ankle clonus. On the right side there was no plantar reflex and no Babinski on either side. The abdominal reflexes were active. Otherwise, there was no change in his condition.

The diagnosis in this case rests between sarcoma of the giant cell type and syphilis. He was given mercurial inunctions up to the point of salivation and a thorough course of iodid of potassium without being benefited. He refused intravenous treatment with arsphenamin. In view of the negative result of the antisyphilitic treatment, it was concluded that the case was probably one of sarcoma of the bone which produced pressure on the brain beneath the site of the tumor.

Cases reported by von Bechterew, Strohmayer and Abraham, in which the lesions were in the posterior, central and lower parietal regions, showed that implication of the brain in these regions, which are the sites of pressure in this case, may give rise to apraxic symptoms. The hemianopsia is more difficult to explain. If the pressure was sufficiently great in the parieto-occipital region it could be explained on the basis of involvement of the optic radiations. Another possibility is that there has been, as a result of a convulsive attack, a cerebral hemorrhage with implication of the optic radiations.

The question of operation arises. If the case is one of giant cell sarcoma, the possibility of the complete removal of the tumor offers the patient a chance of recovery. Without an operation, the case appears to be practically hopeless. I think the patient should undergo an operation to have the tumor removed. I know of no malignant growth of this type which would last as long as this except the giant cell sarcoma, which is the least malignant of all sarcomas. If his diagnosis is correct, the symptoms must be due to pressure by bony growth and not a metastasis, as the latter is not known in giant cell sarcoma.

DISCUSSION

Dr. J. Hendrie Lloyd said he did not see that there was any difficulty in explaining hemianopsia in these cases. An external lesion making deep pressure in that region will be pretty sure to involve the optic radiations and thus cause hemianopsia. Agraphia is usually secondary to word blindness because the faculty of writing depends primarily on the faculty of reading.

Dr. Charles W. Burr said there is a man now at Blockley Hospital with a tumor of the palatal bone, which on his admission had grown upward and affected the base of the brain. He was so weak, not palsied, that he was bedridden. The growth so encroached on the cavity of the mouth that he could scarcely swallow and had to be fed by a spoon with liquids. Speech was impossible. After treatment by radium, the visible size of the growth decreased more than two-thirds. His strength returned so much that he was able to get out of bed, walk and take care of himself. He still has the sarcoma, but life has been made not only bearable but comfortable. Dr. Burr thought it would be worth while for Dr. Rhein to put his patient on radium treatment.

Dr. S. D. Ingham remarked that there was one feature of the case on which he had heard no comment; the absence of choked disk, and to a large extent, of general symptoms of intracranial pressure. It appeared to him that this could be explained by what might be called spontaneous decompression. The erosion of the parietal bone shown in the roentgenogram permitted the large cerebral hernia and prevented any high degree of intracranial pressure.

Dr. John H. W. Rhein said that the tumor seen in his patient was not entirely a bony growth. The skull around the tumor showed some proliferation but in other parts there was a distinct loss of bony tissue. It did not correspond, in his opinion, to the appearances presented by hemicraniosis. It was more likely to be a sarcoma.

ORBICULARIS TIC ATTRIBUTED TO OCULAR IRRITATION BUT DUE TO A RETREAT MECHANISM. Presented by Dr. Tom A. Williams.

A woman, aged 50, was referred by an ophthalmologist because of an obstinate, almost spasmodic contraction of the orbicularis, which had ensued two years before while nursing her sister. It was preceded for a few days by pain in the left eye and for a year by a feeling of uneasiness, which she attributed to faultily fitting spectacles. When she began to screw up her eyes she was chafed by a friend. This caused extra efforts to master the movement, which only made it harder. Lacrimation had lasted a year, though she did not think it was due to the muscular contractions. Several changes of glasses caused no improvement, nor did the constant attention of her husband and friends, and the withdrawal from activities.

Examination revealed sunken eyeballs; active reflexes; increased myotatic reaction; blood pressure: systolic 176, diastolic 118; urine, specific gravity 1.004, with no albumin; blood nitrogen 75 mg. There had been severe headaches the preceding summer and neuralgia and headaches intermittently since the age of 12. Insomnia was distressing. She had always been timid and blushed a great deal; even now the neck flushed during the interrogation.

The most significant feature of the case, however, was that all her life when she wished to shut out an annoying circumstance she had made it a practice to close her eyes when thinking it over. In a crowd at times she would even close her ears to shut out extraneous stimuli which might prevent concentration and adequate reaction to circumstances.

In view of the absence of kidney disease, and in spite of the high blood pressure, and an appearance much older than her years, reeducation was attempted. Various experimental reactions were observed, and she was taught to relax her forehead, and latterly the orbiculares themselves. She was seen every day for two weeks and her psychologic difficulties were rectified in the course of this period. She was made to write out each evening the results of the interview of the day. In addition she was given eliminative baths, compound thyro-ovarian tablets and a low protein diet. Blood pressure eventually fell to systolic 155, diastolic 104. She steadily gained control. She went home in a month, able to read, to go on the streets and to stand considerable stress without the occurrence of the tonic tic which had incapacitated her for two years. There were still occasions, however, when complete control was lacking, but Dr. Williams did not consider it expedient to keep her longer, believing that complete restoration would occur before long.

LACRIMOSITY DISASSOCIATED FROM EMOTIONAL FEELING, WITHOUT APPARENT LESION. Dr. Tom A. Williams.

A clerk, aged 30, had complained of mild headaches for nine months. Three months ago choking and gasping attacks began, with sobbing periods, of which there were about a dozen. Latterly the headache had become constant and more severe at times. He had lost inclination to work, had skin eruptions and an obstinate ulcer of the right foot. There was a history of venereal ulcer ten years ago and he is supposed to have had syphilis in the Philippines, but several Wassermann reactions have been negative.

The reflexes were feeble, especially the right patellar and the left Achilles. There were no other abnormalities. Dr. Williams began provocative injections of arsenic and followed them ten days later by a full dose of neo-arsphenamin. Next day examination of the spinal fluid showed a negative Wassermann and colloidal gold reaction, no increase of albumin, and only two lymphocytes per cubic millimeter. It is perhaps safe, therefore, to exclude meningeal syphilis. The patient insisted that several of the attacks of crying have occurred without the least feeling of sadness or emotion of any kind. In this respect the case is most remarkable, as there are no signs whatever of organic disease, except the preceding symptom and the headache, and there is not the faintest resemblance to the pseudobulbar type of reaction, except in this respect.

A CASE OF PITHIATIC PARAPLEGIA, IN AN EX-SERVICE MAN, ILLUSTRATING RAPID REEDUCATION AT HOME. Dr. Tom A. Williams.

An engineer, aged 37, was seen in Virginia in February, 1920, in consequence of complete paraplegia of three days' onset and ten days' duration. There was a history of paralysis of eight months' duration three years before, after injuring his spine, cured by four months' manipulation. He attributed the present attack to the intense strain of organizing a union. He complained of no pain, only numbness with complete incapacity. Other than a blood pressure of 100 there were no physical abnormalities, all the reflexes being present and normal.

A gradual reeducation was immediately begun in a small cabin in the country in which he was living. One after another the various segments were placed in movement by the patient under strong persuasion, and before Dr. Williams left the patient walked three times around the room. The case is reported, not as a novelty, but to illustrate that even under the most unfavorable conditions rapid reeducation is possible in certain pithiatics.

UNUSUAL SENSORY DISASSOCIATION, DUE TO ORGANIC DISEASE. Dr. Tom A. Williams.

A college student, aged 20, in December, 1918, felt exhausted. In May, 1919, the sight of the left eye became dimmed. In June, supervened fainting attacks with rigidity and spasms, following numbness and dull sensations in the feet, and followed by an unsteady gait. A few months later another attack occurred; she has been unable to walk since that time. When seen in February, 1920, she presented a typical picture of disseminated sclerosis; namely, exaggeration of the deep reflexes, plantar reflexes extensor, abdominal reflexes absent, violent intentional tremor, nystagmus on looking laterally along with slow conjugate deviation, pallor of the left optic disk, especially temporally; poorly maintained contraction of the pupils to light, especially the left, and slow slightly slurred speech. She was mentally clear and there were no emotional disturbances.

The lower limbs were insensible to deep pain and vibration, and a pin prick was scarcely felt as such below the upper third of the thigh. Localization, however, could be made and the temperature sense was retained, not only to heat and cold, but to warmth and coolness. The spinal fluid gave a negative reaction to the colloidal gold and to the Wassermann tests, but there was a slight increase of protein, and seven lymphocytes to the c.mm.

Disassociation of temperature sense and that of pain is unusual except over minute areas in syringomyelia. Head has figured some cases of this kind in a well-known study.

PARALYSIS OF ASSOCIATED UPWARD AND DOWNWARD MOVEMENTS OF THE EYEBALLS. Presented by Dr. Williams B. Cadwalader.

J. W., aged 16, had always been healthy until June, 1919, when he first complained of disturbances of vision. In November, 1919, he was struck by an automobile and knocked down, but there were no evidences of injury other than mild contusions. He seemed well for about a month after the accident and then began to complain of bilateral temporal headache and tremor of his right hand which interfered with writing.

The patient was seen Feb. 24, 1920. The right pupil was larger than the left; neither reacted to light, and they contracted incompletely and sluggishly in accommodation and in convergence. The eyeballs could not be elevated at all above the horizontal plane; they could, however, be moved downward slightly but not more than one third of the normal range. All other ocular movements were normal.

Dr. Deichler reported that there was complete paralysis of the superior rectus muscle and decided paresis of the inferior rectus muscle of each eye. The vessels of the right eye were smaller than normal and the disk was slightly swollen along the nasal border. The left eye was normal.

The right upper limb was distinctly ataxic. His hand writing was tremulous. The biceps and triceps reflexes were slightly increased on the right side, normal on the left. There was no weakness of either upper extremity. Station was normal, but there was distinct ataxia and awkwardness of the right lower limb with a tendency to go toward the right. The patellar and Achilles reflexes were increased on the right, and abortive ankle clonus was present on the right. The plantar reflexes were normal.

One month later the right optic disk showed slight swelling; the left was still normal.

No other abnormalities were found. The headache was not severe and not constant; neither nausea nor vertigo was present. Other cranial nerves were normal; sensation was normal. The patient's general health and intellectual attainments were much better than the average.

Because of the loss of upward movements and weakness of the downward movements of each eye, it is probable that there is a lesion, perhaps a tumor, in the vicinity of the third nerve nucleus of each side or of the corpora quadrigemina. According to the history obtained, the patient must have had some ocular disturbance about a year ago, but the incoordination of the right extremities does not seem to have attracted attention until December, 1919.

DISCUSSION

Dr. J. Hendrie Lloyd asked whether convergence had been tested. He thought that paralysis of convergence had usually been associated with paralysis of upward movement in the reported cases.

Dr. W. B. Cadwalader said that the patient's symptoms were gradually growing more pronounced. Convergence seemed to be paralyzed almost completely.

BILATERAL LENTICULAR DISEASE. Presented by Dr. George Wilson.

A boy, 10 years of age, was born at full term. Labor was easy, and he was not blue at birth. He walked at 5 and had only recently begun to talk. The family history was unimportant.

He was examined because of irregular movements and speech defect.

Examination.—The boy was well nourished. Station was normal. The gait was stiff and punctuated by the general bodily movements which occurred at irregular intervals. The ocular nerves, the pupils, the eyegrounds and fields and hearing were normal. There was no nystagmus. The speech was explosive and dysarthric and dysphagia was present. A spastic grin appeared frequently. All reflexes were presented and prompt, there was no ankle clonus. Plantar stimulation produced plantar flexion although at times extensor response was obtained on the right. The abdominal reflexes were normal. The extremities were hypertonic but not distinctly weak. Sensation in all its forms was normal; there was no astereognosis. The muscles of the face, the upper and lower extremities were all at times involved with a type of movement that seemed to be a combination of athetosis and chorea. The various laboratory tests and the general medical examination were negative.

The movements, dysarthria and dysphagia and hypertonicity of the extremities without marked weakness or positive evidence of pyramidal tract disease led Dr. Wilson to believe that the patient had involvement of the lenticular nuclei.

DISCUSSION

Dr. Charles W. Burr said he had seen this boy at the Children's Hospital, and his impression at that time was that he was suffering from congenital cerebellar disease. The boy was like a miniature edition of Hiram at Blockley; the spasmodic movements of the face, the speech, the athetoid movements and his gait, were all similar.

Dr. W. G. Spiller said that athetosis in the early stage was often overlooked. He had referred many times to a boy who had come to him at 5 years of age in much the same condition. He was now about 20 and in the Home for Incurables, and had developed extreme spasticity and athetosis. Dr. Spiller thought the patient shown probably had a lenticular lesion.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, April 23, 1920

Samuel D. Ingham, M.D., *President*

TWO CASES OF MEDIAN NERVE INJURY. Presented by Dr. N. W. Winkelman.

The first was that of a man, aged 80, who thirty-five years before had had an injury to his left thumb whereby it had been turned backward on the wrist. A few years later he noted, especially in cold weather, a numbness in the palmar aspect of the outer three fingers and the outer side of the fourth finger and also numbness around the nails. Twenty years after the injury pain began in the thumb. At first it was not severe, but within the last year it has become marked, the pain being sharp and more or less constant. Examination showed distinct atrophy of the abductor pollicis, opponens pollicis and part of the flexor brevis pollicis. Motion and power in the hand were good; sensation was but slightly reduced in the proximal part of the thumb though the distal end was hypersensitive; the skin was distinctly shiny. Near the end of the thumb on the anterior aspect was a small pulsating tumor which was evidently an aneurysmal dilatation. Roentgen-ray examination was negative.

The second case was that of an ex-soldier who sustained shrapnel wounds of the right forearm just below the elbow on the radial side. He also showed the same median nerve atrophy of the abductor pollicis, opponens pollicis and part of the flexor brevis pollicis as the patient in the former case but not so marked. Reactions of degeneration were obtained in these muscles. He also had shiny skin and curved nails in the three outer fingers. The sensory disturbances were more marked but were due to involvement of superficial nerves.

DISCUSSION

Dr. Andrew H. Woods mentioned a device for testing the action of the opponens and short abductor of the thumb which he had found useful. The patient was told to hold the hands in a horizontal plane with the palms upward, and then to raise the thumbs into a vertical position "like a tent-pole." The movements of the normal hand were for comparison. With a normal median nerve the vertical position of the thumb can be nearly or completely attained; but any weakness of innervation shows itself by the thumb being carried across the palm by the abductor and lower half of the short flexor, and in a position nearer the palm in proportion to the weakness of the median nerve action.

PERSISTENCE OF NEURALGIC PAIN IN THE TRIGEMINAL REGION AFTER AVULSION OF THE SENSORY ROOT. Presented by Dr. Andrew H. Woods.

Dr. Woods referred to the satisfactory results almost uniformly following avulsion of the sensory root of the trigeminal nerve for neuralgia. The exceptional cases in which pain persisted raised the question as to the existence of other afferent nerve fibers from the face to the central nervous organs. Muscle sense and pressure sense are considered by good authorities to be conducted by the motor root of the trigeminal and by the facial nerves. The glossopharyngeal and vagus may cause confusion in these neuralgic cases when the areas involved are around the ear or in the pharynx.

A patient was presented in whom, after a compound fracture of the right mandible seven years earlier, violent neuralgic pains had continued unabated in spite of neurectomies and avulsion of the sensory root. Angioneurotic edema chiefly of the right half of each lip and the right eyelids had recently occurred, and there was sympathetic palsy of the right eye. An exquisitely sensitive area existed on the oral aspect of the broken jaw, any contact with which started "electric pains" in the lower jaw, referred especially to that sensitive spot. The skin and mucous membranes of the right trigeminal area, except part of that of the ophthalmic branch, did not perceive touch, prick, heat, cold or pressure; but these stimuli usually set off the electric pains. Pressure over the region of the superior cervical ganglion was painful and started the paroxysms. In brief, the essential phenomena of causalgia were present. Dr. Woods felt that there was reason for suspecting the sympathetic nerves in this case and he had recommended that before attempting further intracranial operations an effort be made to dissect off the periarterial plexus of the external carotid artery; or if that failed, to extirpate the superior cervical ganglion.

DISCUSSION

Dr. S. D. Ingham said that this had been a particularly interesting case to him because he had seen a number of cases of causalgia caused by injuries in which the nerve had been completely severed, followed by pain in the distribution of the nerve and excited by peripheral irritation in its distribution. This had been substantiated by operations in which the nerve had not only been exposed, but the stumps had also been resected and still the pain in the distribution of the interrupted nerve persisted. There is a great deal of evidence that there is some side track through which pain impulses can reach the seat of consciousness from tissues, the principal sensory nerve of which has been interrupted. The sympathetic or vegetative nervous system has been shown to possess pain fibers. It is a question whether they should be called sympathetic fibers or whether they are not truly spinal nerves, accompanying the sympathetic and having their cells within the posterior ganglions. Practically all the viscera are supplied with fibers which under certain conditions excite pain, and which must accompany the sympathetic nerves.

A CASE OF HEMIPLEGIA IN A CHILD WITH UNUSUAL SPASMODIC MOVEMENTS. Presented by Dr. C. S. Potts.

History.—Esther, aged 12, was referred to the clinic for nervous diseases of the Polyclinic Hospital by Dr. J. K. Young, on March 8, 1920.

Her mother, father, three sisters and two brothers were living and well. The patient was well until the age of 5, when she was ill with scarlatina in the Municipal Hospital for Contagious Diseases for three months; after this she was paralyzed on the right side and had tremors. Her history while at the Municipal Hospital was not obtainable.

The patient was mentally bright and had reached the sixth grade in school. She was emotional under observation. Her motor disturbance had, however, steadily grown worse, markedly so during the past year; for the past two months she had been unable to attend school. She had never had a convulsion.

Physical Examination.—She was well developed but had never menstruated. The thyroid was somewhat enlarged. The diminution in strength on the right side was not great, but there was marked spasticity, especially when using the limbs. The right leg was 1 inch shorter and the right arm 1½ inches shorter than the left. Below the knee the right leg was 1 inch less in circumference than the left. The arms and thighs were about the same. The knee jerk was greater on the right side. Ankle clonus was not present and the Babinski reflex was absent.

At times while at rest there were athetoid movements of the fingers of the right hand, but when the patient walked the arm was markedly drawn up in flexion and the hand grasped by that of the left side. At the same time, there was marked tonic contraction of the left sternomastoid, and this muscle seemed to be somewhat hypertrophied. Contraction could also be felt in the muscles of the back of the neck on the right side. The head was turned to the right and pulled over until it nearly lay on the right shoulder and slightly backward. The chin was not elevated. At times, when the patient was excited the arm and leg became rigid and fine clonic contractions occurred.

Were the movements of the head organic in origin or hysterical? Dr. Potts believed that there was probably an organic basis but that hysteria played a large part in exaggerating the symptoms. While simulating a torticollis the position of the head was not typical. If organic, would cutting the left spinal accessory and posterior cervical branches on the right side be of benefit?

The case apparently showed that in turning movements of the head the sternomastoid is innervated from the cortex of the same side. The fact that muscular strength was not much impaired and the Babinski reflex absent brings up the question of the possibility of a lenticular lesion.

DISCUSSION

Dr. Charles K. Mills said that it looked to him like a lenticular case. Regarding the forms of sternomastoid spasm or tics, after having had a score of patients operated without benefit, in the course of years, he believed that operation was not desirable. In the case presented by Dr. Potts the child exhibited far more than simple movement of the spinal accessory distribution, for there were more or less involved complex rotary movements of the head and trunk.

Dr. S. D. Ingham said that in the case of paralysis or motor disturbance of cerebral origin outside of acute irritative lesions it was uncommon to have marked spasmodic actions as constant as those exhibited in Dr. Potts' case. Also, bilateral groups of muscles were not commonly affected from cerebral paralysis. Cerebral injuries cause irritation in unilaterally combined groups of muscles. It seemed to Dr. Ingham that the torticollis element of this case was largely of a psychic rather than an organic character and if this were so, operation would probably be worse than useless.

Dr. J. Hendrie Lloyd said that this case partook of the nature of athetoid movements. It was rather peculiar in the violence of the movement in the neck muscles, giving the child a rotary motion. Dr. Potts and others present would remember the classical case of Hiram at the Philadelphia General Hospital, in whom the sternocleido muscles stood out because of overdevelopment from exercise.

Dr. Potts said that he thought there was a psychic element in the case, but there was a question in his mind as to whether there was not an organic basis for it. He felt inclined to believe that probably there was. In regard to the bilateral character of the muscular contractions in turning the head, it must be remembered that this was an associated movement and such muscles may be innervated from the same side, as in lateral movements of the eyes for example. Dr. Spiller before the meeting stated that he believed that the sternomastoid muscle was innervated from the same side of the brain as it appeared to be in this patient. In regard to operation, Dr. Potts thought it offered a chance of relief, but he could not make up his mind to recommend it. The child seemed to be incapacitated for doing any useful work, which before this condition developed she was able to do. She had had large doses of bromids without results. Dr. Potts said he might try atropin; he had written a paper some years ago on the good effects of this drug, as recommended by Leszynsky but he had not thought of it in this case.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Meeting, April 15, 1920

Everett Flood, M.D., *President*

ROENTGEN RAYS OF INFLATED VENTRICLES DISCLOSING A BRAIN TUMOR. Dr. William Jason Mixter.

The first case I present is one in which I consider I made a definite mistake, with a fatal result. It was one of unlocalized brain tumor in a child, presumably cerebellar, with internal hydrocephalus, an almost decerebrate animal according to symptoms. The roentgen rays did not reveal anything definite in the way of a tumor although a shadow could be seen in the center of the cranium. Roentgenograms made after the cerebrospinal fluid in the ventricles had been replaced by air under atmospheric pressure showed a different picture, for a large tumor mass presented itself distinctly. This injection of the ventricles has been done by Dandy a considerable number of times, I think without as yet a fatal result. Unfortunately, my first case was a fatality, the child dying within twenty-four hours. My feeling is that the mistake was made in letting in too much air. I think death probably was due in part at least to change in the pressure of the cerebrospinal fluid. At necropsy a large tumor of the pineal gland was found.

Surgical Treatment of Meningitis.—Some four years ago I operated on a patient with cerebrospinal meningitis and tapped the ventricles, replacing the infected fluid with serum. The patient received this treatment six times and made a practically uneventful convalescence. Previous to the intraventricular treatment he had had numerous spinal treatments and the cerebrospinal fluid by lumbar puncture was negative. The case showed a mild grade of choked disk and definite though mild symptoms of increased cranial tension. The case was reported by Dr. Hoch in the *Boston Medical and Surgical Journal.*

When in France I was much interested in a case of cerebrospinal meningitis in a nurse who finally died of a chronic type of this disease three months after her original infection, having shown no evidence of increased tension or definite evidence of infection, involving the upper meninges. At necropsy her ventricles were found to be filled with a turbid fluid showing meningococci in large numbers. If I remember rightly there was practically no infection in the tissues of the lower spine. She did, however, have an abscess in the spinal cord. She was not given this ventricular treatment.

A short time ago Dr. Ayer asked me to see a patient with cerebrospinal meningitis whom he had been treating for some days, first with lumbar puncture, later with cisterna puncture, who had at first responded well to treatment and after a time had not responded although the cerebrospinal fluid by lumbar puncture was negative. Exploration of the ventricles showed turbid fluid. The patient was treated twice by injecting into the ventricles about 40 c.c. of serum, and she is now ready to leave the hospital.

These cases to my mind are rather significant. In patients with cerebrospinal meningitis who are not reacting to treatment but are still having temperature and especially an increased white count notwithstanding the absence of organisms in the cerebrospinal fluid, I think that ventricle puncture, even if only for diagnostic purposes, is indicated. It is not, as a rule, a difficult operation.

DISCUSSION

Dr. James B. Ayer: It was my privilege last year to do a great deal of work on experimental meningitis. In the animals on which we experimented we found that within twenty-four hours after meningitis had commenced the greater number showed also ventricular infection. It is not known whether or not this early and frequent infection of the ventricles is true of man, but owing to the greater depth of the velum interpositum and velum medullare in man, by which it is presumed infection reaches the ventricles, it is probably less frequent and less early than in the experimental cat or rabbit. However, there is reason to think that the organisms usually reach the human ventricle provided the meningitis lasts long enough. In this case, the meningitis had lasted for three weeks in spite of vigorous treatment with a known effective serum. If, as in the animal, it is possible for ventricular infection to occur in twenty-four hours, or several days, it is unreasonable to treat patients indefinitely by lumbar puncture, or even by the more direct cistern puncture. The ventricles must be reached directly. What shall be our guide as to when ventricular exploration shall be made? If after a reasonable amount of treatment by the subarachnoid route with a serum of known efficacy in the case at hand, either (1) the organisms persist in the spinal fluid or (2) the organisms disappear, but symptoms of acute meningitis persist, it is probably wise to investigate the ventricles.

THE ANATOMIC IMPLICATIONS OF INTROSPECTIVE PSYCHOLOGY. Dr. Harold I. Gosline.

Introspective psychology is not an "arm-chair" psychology but was developed and is being added to by laboratory methods. It had its inception in the first psychologic laboratory established by Wundt in 1876. Corresponding to this beginning and to its aims, this form of psychology is built up like any other science. It makes certain fundamental assumptions its postulates and builds around these, assuming them to be true till some experience is reached which is not in accord with the postulates. Until then the postulates must stand as the truth.

On the basis of this psychology an analysis of personality is presented which reduces the complex mental functions, inner states, activities, ideas and perceptions into simple mental processes, sensations, associations, reactions and inhibitions.

If this idea of introspective psychology is the truth, the author supposes that it can be made the basis for some conceptions of nervous system anatomy, which were then presented.

THE STATE INFIRMARY. Dr. J. H. Nichols.

Dr. Nichols read a paper on the State Infirmary at Tewksbury, Mass., giving the history and development of that institution from the time of its inception, with a description of the present scope of its activities.

Book Reviews

SHELL-SHOCK AND OTHER NEUROPSYCHIATRIC PROBLEMS. Presented in Five Hundred and Eighty-Nine Case Histories, from the War Literature, 1914-1918. By E. E. Southard, M.D., Sc.D. Bibliography by Norman Fenton, S.B., A.M., Sergeant, Medical Corps, U. S. Army; and an Introduction by Charles K. Mills, M.D., LL.D., Emeritus Professor of Neurology, University of Pennsylvania. Cloth. Price, $10. Pp. 982, with illustrations. Boston: W. M. Leonard, 1919.

Whether or no the participants in the World War will have the acumen to profit by the lessons taught by that great conflict, is one of the vital questions of the day. Dr. Southard in the present work tried to contribute his share to an affirmative answer. He lived to see his book in print, but not to view the harvest sprung from his sowing.

The author calls the work a compilation. So it is; but it is more. Not only are there case records (589 of them) from the literature of many countries covering every phase of war neuroses and psychoses, but there is an epicrisis that gives an excellent digest of the enormous clinical material: The discussion is psychologic and psychiatric, philosophic and practical, expounding principles and also indicating the clinical steps essential to avoidance of errors. Treatment and results are adequately covered.

Besides being an altogether unusual exposition of the nervous disorders of the great war, it should be most valuable to those engaged in reconstruction work. The physician engaged in civil practice only may also learn priceless lessons from this book.

The first 265 pages (Section A) contain clinical records (from the literature) representing eight groups of disorders or defects that influence or complicate war neuroses or psychoses. These are (1) those patients in whom syphilis possibly or certainly plays a rôle, (2) the feeble-minded group, (3) the epileptic group, (4) the alcohol-drug-poison group, (5) the group with focal brain lesion, (6) the symptomatic group, (7) the presenile group (no cases) and (8) the dementia praecox group.

Section B (251 pages) in the same way covers shell-shock, its nature and causes; Section C (142 pages), shell-shock, diagnosis; Section D (156 pages), shell-shock, treatment and results; and Section E (72 pages) epicrisis. There are over 2,000 bibliographic references.

We can say with Dr. Mills in his able introduction that "in this wonderful collection of Dr. Southard is to be seen an epitome of war neurology not elsewhere to be found." The epicrisis is an interesting discussion of many phases of the subject. It is also inherently instructive. It abounds in sound direct and inferential advice to physician, government and army. No one can carefully read it, whether he agrees with the author or not, without acquiring a broad conception of war neuroses and their complications. If the physician has a broad conception of war neuroses, he has a fairly good grasp of peace psychoneuroses—a consummation devoutly to be wished. Furthermore, every intelligent reader will be interested in, instructed and entertained by the Southardian philosophic and linguistic agility.

However, it should be understood that in no sense is this work a textbook or systematic treatise. It is an exposition of many things, with a number of sage comments, wise admonitions and critical suggestions by a capable cicerone.

THE HYSTERIA OF LADY MACBETH. By Isador H. Coriat, M.D. Moffat, Yard and Company, New York, 1912. Pp. 92.

This is an interesting little essay on Lady Macbeth. The book is apparently intended for the lay public, for in the first, second and third chapters there is an exposition of the general principles of psychanalysis and its reference to literature. In brief the author states that "Lady Macbeth is a typical case of hysteria; her ambition is merely a sublimation of a repressed sexual impulse, the desire for a child based on the memory of a child long since dead. In fact an analysis of the sleep-walking scene demonstrates that it is neither genuine sleep nor the pricklings of a guilty conscience, but a clear case of pathological somnambulism, a genuine disintegration of the personality."

According to the author, Macbeth was an epileptic, which explains one phase of his criminality. Coriat says that "the four great tragedies of Shakespeare have sexual problems as their central motive. The father problem appears in Lear and Hamlet, the evolution of a jealousy complex in Othello and the theme of childlessness in Macbeth." The author goes on to say that Shakespeare was not a scientist, but he was able intuitively to demonstrate the mental mechanism of his characters. Whatever the reader might think of this point of view, the book is interesting and worth reading.

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ELEMENTS OF PREDISPOSITION AND DETERMINING CAUSES OF SECONDARY DISORDERS IN PSYCHONEUROTIC ACROPARALYSIS RESULTING FROM THE WAR: "THE DYSKINETIC SYNDROME"

GUSTAVE ROUSSY, M.D.

PARIS

Among the numerous nervous disorders brought to light by the war, there is a syndrome with peculiar clinical manifestations which has impressed the French neurologists (Meige, Pierre Marie, Foix, Babinski, Roussy, Boisseau and d'Oelsnitz). This syndrome is characterized by contractures resulting in fixation of the larger articulations (hips, shoulders, elbows and knees) in defective postures, and by motor difficulties of a special type—contraction, paralysis or an association of both—localized in the distal ends of the limbs and producing a condition described by the French as "main figée" (palsied stiff hand) and "pied bot réflexe" (reflex club foot).

Following a nervous shock caused by the bursting of a shell in close proximity, or traumatism, often slight, or certain other causes, a state of impotency, dependent on a more or less well developed pithiatic character, becomes manifest. At the end of a period of time, which varies in the different cases, the influence of immobility or defective function, somewhat conditional on certain predispositions (circulatory, etc.) leads to secondary organic accidents, lasting as long as the immobility itself; these, however, gradually decrease and eventually disappear after an indefinite length of time, if the normal movements are restored.

The question, therefore, is no longer that of mere pithiatic accidents, but really of hystero-organic associations. Consequently two elements of the problem to be considered are: (1) the chief motor disorder, (2) the secondary disorders (vasomotor, trophic, etc.), which develop later.

I shall attempt at this time, without reference to the extended series of reports that have been published in France on this subject, to recapitulate the different signs and symptoms which constitute this syndrome.

MOTOR DISORDER

Motor disorder (contraction or paralysis) has long been considered obstinate when treated by psychotherapy. I have demonstrated, however, with Boisseau and d'Oelsnitz that it usually assumes the characteristics of a pithiatic accident, namely, it can be made to recur or disappear suddenly by persuasion. I also stated in previous publications that the posture, dependent on paralysis or contracture, does not seem at first sight to be capable of reproduction on the normal side by the patient. However, it is possible to demonstrate that a state of individual articular or musculotendinous laxity may be present, enabling the patient to assume attitudes not normally possible.

SECONDARY DISORDERS

Secondary disorders are the clinical characteristics of the organic accidents which Babinski and Froment first observed and minutely described. We shall choose, as a standard, those accidents that are described as "stiffened hands," in which the complete array of signs and symptoms constituting this syndrome may be observed.

Vasomotor and Thermic Disorders.—The skin of the hand and sometimes of the forearm as well, is cyanotic, the color varying from bluish or purple to salmon red. The surface temperature may be from 2 to 6 degrees (centigrade) less than that of the normal hand. By means of a Pachon sphygmomanometer it is easy to discern that the arterial oscillations, especially during cold weather, are less in amplitude on the affected side (Babinski and Heitz). These authors have shown, moreover, that after a bath of five minutes' duration at 40 C., the affected hand is warmer, and the arterial oscillations of greater amplitude than on the healthy side. Therefore arterial lesions may be excluded as a cause of the cyanosis. Moreover, I have observed, with Boisseau and d'Oelsnitz, that sudden restoration of active movements after treatment produced the same result (disconnecting test). Furthermore, I have pointed out that on the healthy side of such patients, there were more or less obvious vasomotor disturbances, showing that they, possibly on account of a congenital predisposition, have vasomotor disturbance. This fact proves that the patients, possibly on account of a congenital predisposition, do not possess a vasomotor system which functions normally, and that, therefore, the marked changes on the affected side are more easily produced by factors dependent on immobility than in normal persons.

Mechanical Overexcitability of the Muscles.—By tapping the muscles of the hand or arm of the affected side with the finger, the muscular contraction produced is stronger and of longer duration than on the healthy side. The electrical excitability of the muscles and nerves may be modified likewise, although a reaction of degeneration is never present.

Disorders of Sweat-Secretion.—Hyperhidrosis (over-sudation) is often present on the affected side, sometimes so marked as to produce sudamina. I have pointed out that this disorder existed in patients in whom a tendency to excessive sweating was also present on the normal side.

Trophic Disturbances.—These are often well developed. The skin becomes thin; smooth and glossy. The wrinkles are obliterated. When the vicious attitudes are of long duration, genuine cutaneous retractions may occur. In not a few cases the skin is infiltrated and appears "succulent." The nails become hard, brittle and show transverse or longitudinal striations. Occasionally they are convex, hippocratic and deformed. They often grow faster than on the normal side. The hair is longer and thicker than on normal side, or on the contrary, broken, smaller and less abundant.

Some muscular atrophy, generally slight, may occur. Occasionally, however, the circumference of the arm may be from 4 to 6 cm. less than that of the normal side. Tendinous contractures are often present in long standing cases. The phalangeal and metacarpophalangeal articulations are frequently stiff and painful and at times even fibro-ankylosis is present. Nodular deformities, such as are found in arthritis, may occur. Even the bones may be so altered that decalcification and osteoporosis is revealed by roentgen-ray examination.

Modification of the Tendinous and Cutaneous Reflexes.—Babinski and Froment have shown that the tendon reflexes may be exaggerated on the affected side and that this increased excitability becomes even more pronounced when the patient is anesthetized by chloroform. With Boisseau and d'Oelsnitz, I have demonstrated for the first time the relation between this hyperreflectivity and muscular atrophy. The ankle reflexes are diminished (Vincent) and plantar response by plantar stimulation may be abolished (Babinski and Froment). These modifications occur in instances in which cyanosis and hypothermia are prominent features. They disappear as soon as the limb becomes warm.

Muscular Hypotonicity.—This may be so marked in certain instances that the palmar surfaces of the fingers may touch the anterior surface of the forearm.

The association of the motor disorders with the secondary disorders just described forms a peculiar clinical syndrome, whose localization in divers segments of the limbs gives rise to a series of psychoneurotic accidents which cannot be completely detailed within the limits of this article. I shall, however, mention a few of the more frequent ones.

I have chosen as the typical case the "stiffened hand" because in such instances secondary accidents are not only common and as a rule well developed, but also because this condition has been studied with special care. In foot palsies and acrocontractures (psychoneurotic club-foot, clutched toes) and in psychoneurotic contractures of the elbows, knees, hips and shoulders, these secondary phenomena are often present. They may occur not only in palsies and contractures of segments of the limbs, but also in some instances of monoplegia or pithiatic paralysis.

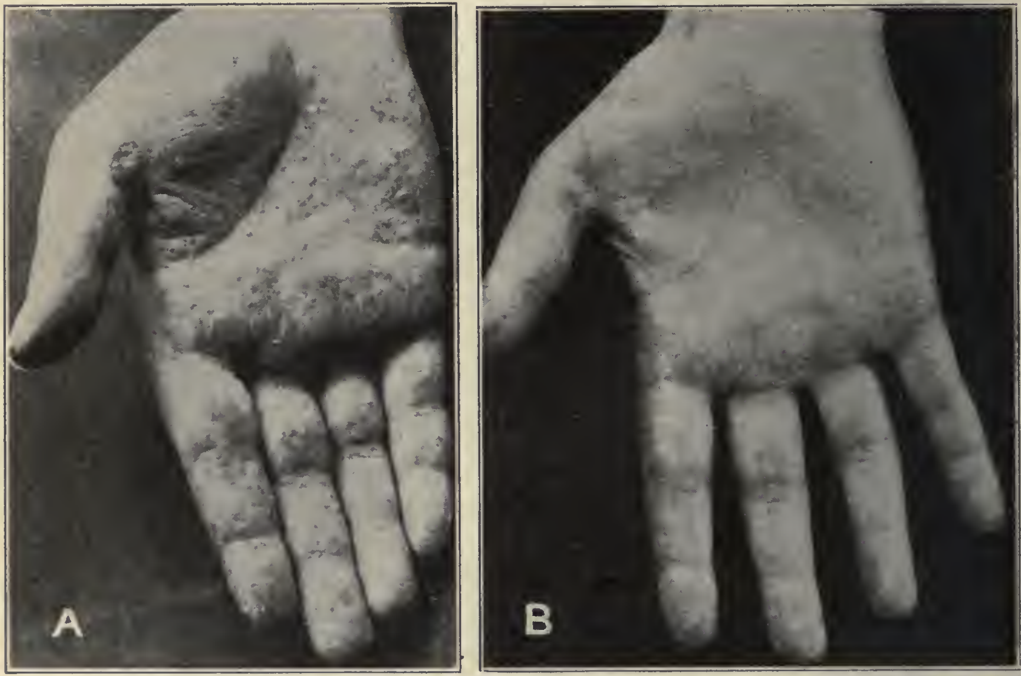

Fig. 1.—Hand from a case of contracture of the elbow and difficulties of flexion of the first phalanx of the third and fourth fingers, with fibrous formations and limited function, eleven months after the injury in the collapse of a dugout; *A*, showing numerous sudamina caused by hyperhidrosis; *B*, disappearance of cutaneous disturbances, including hyperhidrosis, after the recovery of motor function.

Nature of Vasomotor and Trophic Disturbances.—In what group do the disorders just described belong? Are they hysterical or organic? These questions, often discussed during the war, have not been unanimously and finally solved. Without entering into details, I shall mention briefly the principal theories that have been advanced in France as an explanation of their pathogenesis.

The Reflex Theory: According to Babinski and Froment, these accidents fall into a special group "intermediate between the strictly organic and the hysterical disorders." They attribute the origin of these disorders to reflex causes and insist, in their later reports, on the frequency of the association and the progressively predominant influence of the hysterical element, sometimes amounting to the complete replacement of the pithiatic element.

The Dynamogenic Theory: Claude and Lhermitte admit that in the beginning of the functional motor disturbances, an organic lesion (slight wound) may, in certain instances, operate by a reflex mechanism. But—and here they differ from the former theory—this reflex mechanism (originally causal?) plays only a short and transitory rôle.

If treated properly, the disorders will disappear without leaving any residuum (circulatory, articular, trophic), but if not so treated, the conditions become fixed by a psychic mechanism, and their constant presence produces dynamic modifications of the nervous centers, as shown by the study of motor, sensory and reflex phenomena during the narcotic state.

The Dyskinetic Theory: (*The Immobility or Defective Action Syndrome.*—Under this title, in cooperation with Boïsseau and d'Oelsnitz, I have grouped the ideas that we have expounded before. The basis of our conception, supported by and founded on the careful clinical observations of more than 2,000 cases of war psychoneuroses, may be stated thus:

1. The motor disorder, contraction or paralysis, is the principal and primary element of the syndrome; it is also pithiatic in character and consequently capable of reproduction at the will of the patient. It is liable to disappear suddenly under the influence of persuasion if this method is employed under favorable conditions.

2. The associated disorders, which are commonly but not constantly present, are variable in character and very persistent. They are, according to Babinski's classic conceptions, comparable in every way to disorders admittedly hysterical in character. They are, furthermore, functional accidents that have suddenly appeared in patients predisposed to pithiatic motor disturbances.

Can our conceptions of their pathogenesis be made to agree with the facts observed? We believe they can and I shall therefore recapitulate certain arguments in support of our opinion.

It is well known that the secondary disturbances do not appear indifferently in every instance of pithiatic motor disorder. What, therefore, are the causes and laws on which this selective association depends? As the result of our investigations we are inclined to believe that the origin of these secondary disorders is doubly conditional and dependent: (1) on elements of constitutional or acquired predisposition antedating the pithiatic motor disturbances, and, (2) on determining causes operating for a longer or shorter time on an adequate predisposition and producing completely the array of secondary disorders.

PREDISPOSITION

The systematic observation of the healthy half of the body of patients affected by neurotic disorders has led to certain interesting conclusions. The most typical and complete example is that of the "main figée." Its clinical characteristics are: hypothermia and cyanosis are present; oscillometry demonstrates more or less marked diminution of the arterial oscillatory amplitude; sudoral disorders are present; the reflexes are modified; and there are deformities, grotesque in appearance and apparently not possibly produced by the will or by an hysterical condition. We have constantly attempted to discover whether these various alterations were not also present in a larval or latent state on the unaffected side, and our efforts in this direction have not been unrewarded.

Repeated examinations of the minor alterations of function on the healthy side have demonstrated that the same tendencies are present but of lesser degree. Therefore, the same predisposition to pithiatic disturbances has been identified in both limbs but at different functional levels. I shall now describe these phenomena and later discuss their significance.

The Circulatory Phenomena, Microsphygmia.—Oscillometric investigation of the numerous cases of acromotor disorder has disclosed that the healthy side of the patient is in a condition of vasoconstriction not so marked as on the affected side, but definite as compared with the results obtained on normal subjects. This condition is sometimes appreciable by sight due to the acrocyanosis. We have verified this observation not only by repeated readings with the Pachon oscillometer, but also by graphic records or "oscillograms." It is apparent, therefore, that particular stress should be placed on the existence of bilateral microsphygmia in patients with "main figée" and other pithiatic disturbances.

The Sudoral Phenomena.—The systematic studies of the healthy side under different conditions have demonstrated certain other tendencies. If a hand is perspiring so abundantly that it seems glossy and beads of sweat appear on the palmar surface, the same tendency to hyperhidrosis, less marked but still abnormal, will be observed on the other hand.

Articulo-Musculo-Tendinous Laxity.—If extreme hyperextension of the fingers is present, apparently not produced by the will or even by an hysterical disorder of the affected side, decidedly abnormal musculo-tendinous laxity will be found on the healthy side. It is under these conditions that antalgic attitudes, often voluntarily fixed, are apt to occur. The deformities so produced at first sight seem to exceed the limits of purely hysterical disturbances.

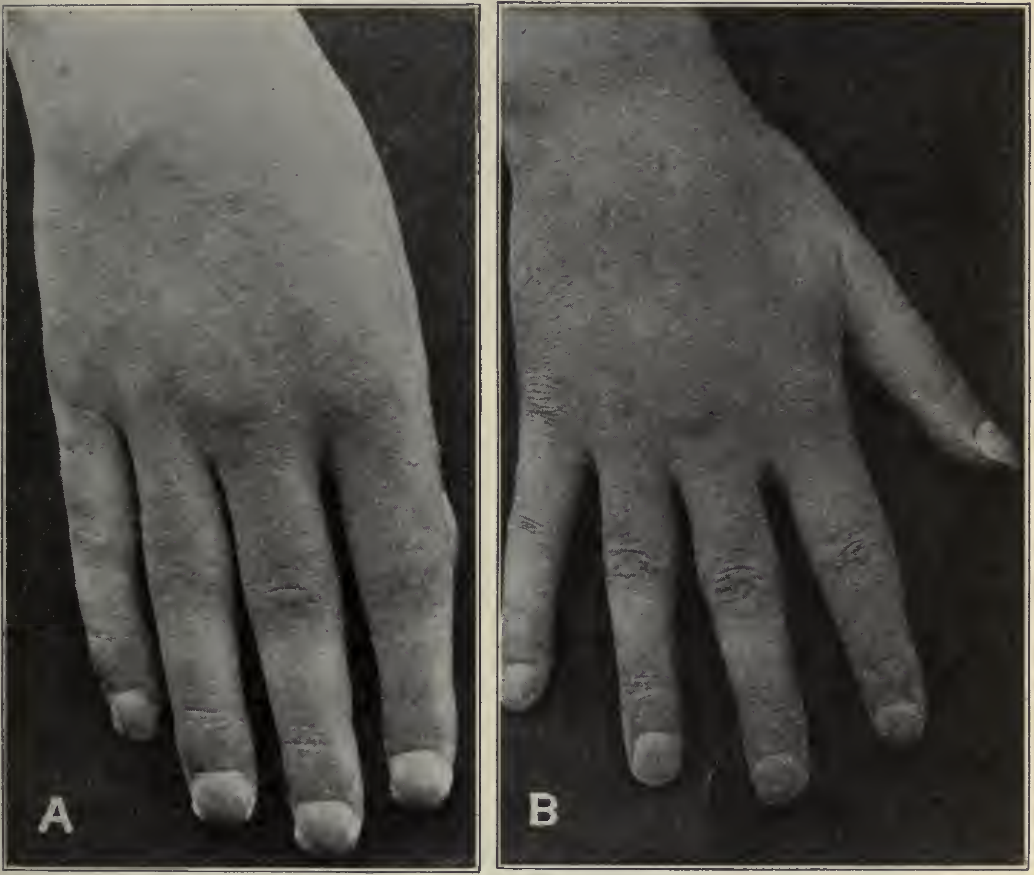

Fig. 2.—Right hand paralyzed (main figée), thirty-five months after penetrating gunshot wound of the forearm; *A,* showing trophic disorder of the nails (longitudinal incurvation) and of the skin (glossy appearance) with disappearance of the wrinkles around the knuckles; *B,* reappearance of the wrinkles after several weeks, during which the patient has regained the use of his hand.

ARTICULAR DISORDERS

Why are certain persons subject to early articular stiffness while others show no signs of the disturbance even after a long period of immobility? Although immobility is necessary for the production of articular disorders, alone it is not a sufficient cause. A variable association of predisposing causes is required. In certain instances long suppuration appears to promote articular reactions, in others former articulatory disturbances bring up the question of a diathetic predisposition. Immobility and predisposition therefore appear to be necessary and sufficient for the origination and development of the articular symptoms.

TENDINOUS HYPERREFLECTIVITY

Should clinical observation detect a state of excessive tendinous reflectivity, the healthy side will usually reveal a tendency toward strong reflex reactions. This has been proved daily during clinical investigations. How may we explain this amplification of the tendinous reflexes? It is noteworthy, as stated above, that the coexistence of hyperreflectivity and muscular atrophy has frequently been observed. Moreover, in a number of patients a slow but progressive diminution of tendinous hyperreflectivity has been observed coincident with the amelioration of the motor disorder and paralleling the resumption of normal function, and consequent subsidence of muscular atrophy in the affected limb.

I believe, therefore (taking into account the results obtained by Babinski and Froment from the study of the reflexes in a prolonged narcotic state, and without rejecting Claude and Lhermitte's ingenious hypothesis of the dynamogenic action operating through the long inactivity of the limb on the medullary centers), that another hypothesis can be advanced. Cannot the reflex modifications be caused (in certain cases, and to a greater or less degree) not by an anatomic or dynamic alteration of the center (spinal cord) of the reflex arc (conductors, nerves) but by an affection localized at the starting point of the arc —the muscles? In this instance the tendinous hyperreflectivity might be connected with the anatomic (histochemical) modifications and thus with the physiologic modifications of the muscle fiber itself (myofibrilla or sarcoplasm).

This consideration of the abnormalities observed on the healthy side in hysterical patients has made it possible to perceive dimly, in their larval state, most of the disorders of the affected side. But are alterations found that antedate the morbid disturbances (genuine elements of predisposition) rather than the direct influence of the affected side on the normal side? I believe so. The disorders of the healthy side cannot, when subjected to early and repeated observation, be explained by the laws of symmetry; and moreover we usually notice that, according to their presence or absence, the same neuropathic motor troubles will or will not bring about morbid aggravations on the side affected.

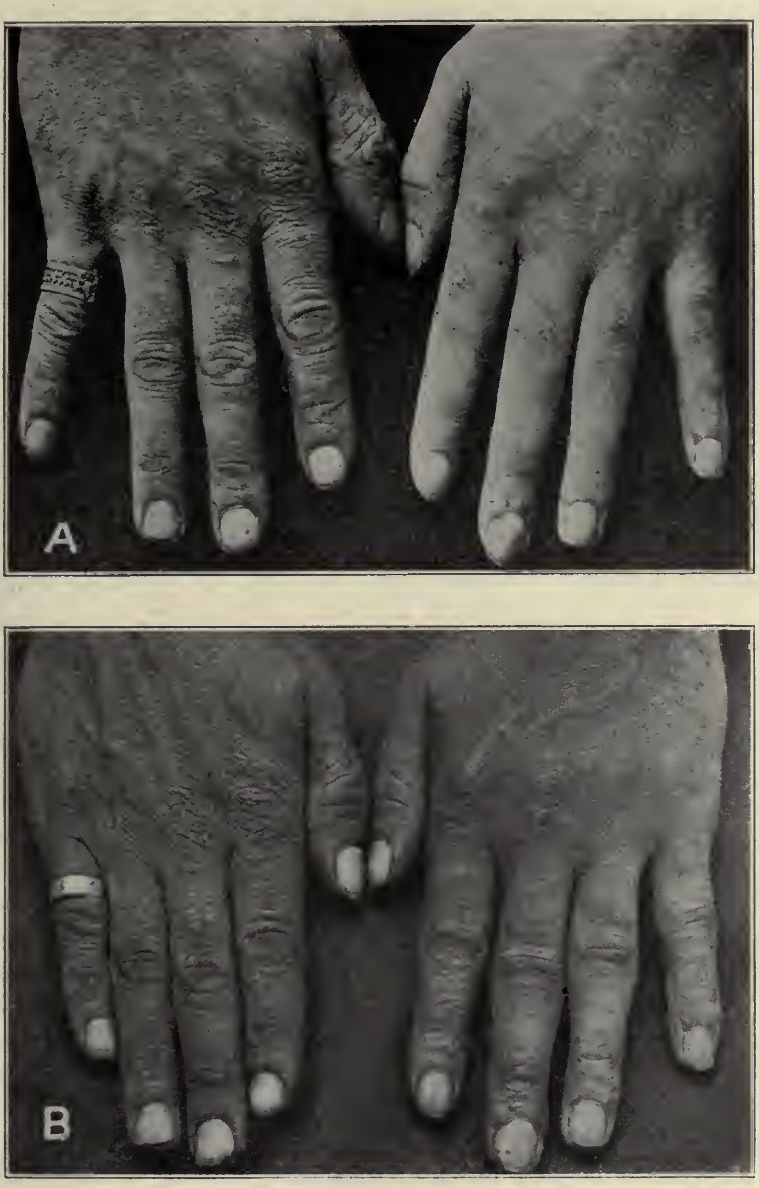

Fig. 3.—Paralyzed left hand (in extension), twenty-seven months after a fistulous bullet wound of the forearm, with reflex disorders; *A*, showing the trophic involvement of the skin and nails of the left hand, as is clearly discernible by comparison with the right hand; *B*, reappearance of the wrinkles around the knuckles following marked improvement several weeks after the restoration of function.

The preexistence, therefore, of bilateral and symmetrical disorders dependent on predisposition appears to be the most easily admissible hypothesis to explain our observations. Hence, the most plausible hypothesis is that the full development of the secondary syndrome observed on the affected side is only an abnormal amplification of preexisting tendencies observed on the healthy side.

But what are the circumstances that have brought about such an amplification or accentuation and have thereby converted a manifestation that is physiologic into a disorder obviously pathologic?

DETERMINING CAUSES

We may now observe the intervention of two essential factors: the influence of immobility or defective function and the influence of the pyschic state of the patient.

The Influence of Immobility or Defective Function.—I believe that antalgic immobility through psychic inertia, following traumatism, as well as immobility obtained by compression in any fracture—reducing apparatus—is enough to aggravate the preexisting tendencies and cause secondary disorders to appear; and that later, the defective function of the limb or limb fragment promotes their progressive evolution and development.

Thus the different disorders will occur in this chronological order: the caloric and vasomotor conditions with their sequelae (mechanical hyperexcitability, modifications of the electric reactions); the trophic disturbances, amyotrophia (with consequent hyperreflectivity), articular fibro-ankylosis and musculotendinous retraction. If immobility alone does not generally suffice to give rise to circulatory disorders which preexist, it may, when associated with previous vasoconstriction, occasion them. Thus immobility and vasoconstriction, though of independent origin, are factors which undergo a parallel evolution and add their effects to produce the syndrome in its symptomatic whole.

My conception of the mechanism of its production is, therefore, that the bilateral acrovasomotor disorders are themselves dependent, even before any traumatic or dyskinetic element appears, on a certain degree of irregularity in the vascular sympathetic system. They betray a deficiency in the thermic and vasomotor functions against which the patients are obliged to react by constant straining of the muscles. The intensity and frequency of the movements make up for the retardation of the circulation by exciting the inadequate functions of the sympathetic system. Under these circumstances, when a limb, or rather a limb end, in which such troubles are found, lies motionless under direct (antalgic) or indirect (pithiatic) influence of some local traumatism, what will occur? "The organs"— to quote Morat and

Doyon—"produce, through the medium of their sensory nerves, the vasomotor phenomena destined to equalize the circulation to the need occasioned by the effort."

The muscular inactivity will, therefore, increase the inhibition of an already inefficient sympathetic system. The longer this inactivity lasts, the more accustomed the sympathetic system becomes to the new state of things and it acquires a decided tendency to lose completely its normal regulatory functions. What happens if the motor functions of the limb are made to reappear?

Passive mobility will only modify for a time, and in a very limited manner, the vasomotor conditions; this is accounted for by the fact that passive motion is not a revival of the muscular functions but only a false appearance of the physiologic action of the muscles. Consequently there is no reason to expect it to influence the sympathetic centers in any important or lasting manner. On the contrary, the recurrence of active movements causes, as has been shown, an intense modification with remarkable improvement of the vascular disorders. If the result does not prove complete and final, if it shows a tendency to stabilize gradually, it is, in my opinion, because the sympathetic system of the limb has grown accustomed to muscular inactivity and because the adaptation to renewed activity requires regular and continual exercise of the muscles in their normal physiologic function.

It is therefore my opinion that immobility leads to the appearance of secondary disorders in predisposed persons. It is the suppression or the defectiveness of their functions that allows the disorders to persist. Restoration of activity leads to improvement and physiologically normal exercise is likely to cause the disorder to disappear.

Necessity of a Special Mental State.—A persistent, systematic and even unnatural cessation of function is necessary to produce such disorders. The patient apparently does all in his power to lose the use of his limb or limb fragment. The patients are persons with a special turn of mind, whose will is deficient or more or less perverted, reminding us in many particulars of that of patients injured in industrial accidents. This state of mind is an essential etiologic factor necessary for the growth and development of the disorders considered. It enables them to appear, explains their characteristics and furnishes rules to be followed in their treatment. The mental state must be always taken into serious consideration in deciding the prognosis and treatment of a psychoneuropathic accident.

SUMMARY

After an investigation of more than 2,000 psychoneurotic disorders, these conclusions appear to be justifiable:

1. To enable the appearance of vasomotor and trophic disorders, during the course of acrocontractural and acroparalytic manifestations, three conditions are necessary: (*a*) Elements of predisposition and among them circulatory troubles, (*b*) immobility or defective function, and, (*c*) a special mental state.

2. The circulatory caloric and trophic troubles are only manifestations of a secondary class arising from a primary condition of neuropathic nature, paralysis or contracture.

I have therefore, proposed to designate the whole series of disorders constituting this syndrome "the dyskinetic syndrome, or syndrome of defective function."

PERIPHERAL NERVE TOPOGRAPHY

SEVENTY-SEVEN OBSERVATIONS OF ELECTRICAL STIMULATION OF NORMAL AND DISEASED PERIPHERAL NERVES

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1. Introduction
2. Technic
3. Material
4. Levels of Stimulation
5. Electric Phenomena
6. Plexus Formations in Nerves, Fasciculi and Fibers
7. Preponderance of Reaction in Proximal Muscles
8. Straight Course of Motor Fasciculi
9. Findings in Individual Nerves and Their Branches
10. Case Reports and Charts

INTRODUCTION

The large number of injuries to peripheral nerves which occurred during the world war centered the attention of many on the details of their anatomy. It became important in considering partial lesions to know the functions of the various fasciculi that made up a nerve. Clinical examination often indicated an incomplete lesion, and a study of the course of the bullet or piece of shrapnel showed that one or another aspect of the nerve trunk had been the main site of the injury. In other cases compression by scar tissue affected the nerve function. When the case reached the stage at which operation was performed, the question sometimes arose—What relation is borne by the clinical to the operative findings? Does an external lateral neuroma mean that certain muscles must be paralyzed, and does this coincide with the clinical findings? Figure 1 from Dustin's[1] paper gives a graphic idea of this problem. In addition to these questions, others of more theoretical value arose. The whole problem of the anatomic basis of physiologic facts or theories relating to sensation and the conduction of stimuli to muscles in the peripheral nerve made its appearance.

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[1] Dustin: La fasciculation des nerfs, in Ambulance de "L'Ocean," **2**:pt. 1, 135 (July) 1918.

All this resolves itself into the question—What physiologic significance has each intraneural bundle of fibers or fasciculus? Is it motor, and if so, what muscle does it innervate? Are sensory fibers included in motor fasciculi and how do they reach them? Which fasciculi are purely sensory? When segments contribute to a nerve, where do their various fibers rearrange themselves? The questions multiplied. It became clear that we had little knowledge of the physiologic aspect of the numerous fiber tracts (fasciculi) contained in the various nerves. Compared to our knowledge of spinal cord tracts, that of peripheral nerve tracts was extremely meager.

Stoffel,[2] in 1911, did some thorough work on this subject. By gross dissection, he succeeded in separating a large number of fasciculi, in determining the gross appearance on cross section of many of the larger nerve trunks and in correlating these pictures with the functions of the various fasciculi appearing in them. He correlated the transverse as well as the longitudinal pictures in terms of the location and function of the fasciculi.

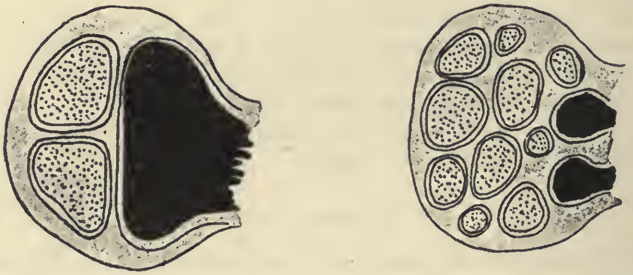

Fig. 1.—Involvement of fasciculi from a lateral neuroma (from Dustin).

The next work was done in France and concerned war injuries. Marie[3] and his co-workers, by means of electrical stimulation of nerves exposed at operation, confirmed some of the conclusions of Stoffel and added new facts. They correlated the cross section work of Stoffel with the findings they obtained by stimulation of the circumference of the nerves and found that they agreed in many instances.

In England, Langley and Hashimoto,[4] using a third method—dissection of macerated nerve trunks—made contributions to the subject, mainly in regard to the interlacing of the fibers of various fasciculi, i. e., *internal nerve plexuses.*

Dustin[1] of Brussels, in July, 1918, did a large piece of work by gross dissection and by correlating the appearance of successive cross-sections of the nerves. His article contains illustrations showing the cross sectional appearance of the various parts of the brachial plexus and the median nerve; but the work is purely anatomical and, though of importance, led to no correlation with the functions of the fasciculi he describes.

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[2] Stoffel and Vulpius: Orthopēdische Operationslehre, 1911.

[3] Marie, Meige, Gosset and Pascals: Les localizations Motrices dans les nerfs peripheriques, Bull. de l' Acad. de méd., Par. **74**:798, (Dec. 28) 1915.

[4] Langley and Hashimoto: J. Physiol. **51**:318, 1917.

Therefore, by the end of 1918, considerable work of various kinds had been done. The present investigation is a continuation of that of Marie, and has the same aim in view, namely, to determine, by application of a faradic current to the circumference of the nerve, the location and function of the fasciculi that lie beneath.

TECHNIC

This article is based on work done on nerves, normal and abnormal, exposed at operation in the U. S. army hospitals at Cape May, N. J. and Fox Hill, N. Y., in 1919. The difficulties of investigation are not many if care is taken to follow a few simple procedures.

Orientation of the Nerve.—The various anatomic aspects of the nerves must be properly labeled to permit ready recognition at any time. The details of this and of the technic at operation are minutely described in a previous article.[5]

MATERIAL

Of the cases in which an attempt was made to localize bundles, only those that gave a positive result are included. Approximately 25 per cent. of the cases tried gave a negative result, due to complete functional interruption of the entire nerve. The positive cases, fifty-eight in number, on which seventy-seven observations were made, are charted in Table 1.

The positive cases reported by Marie, Meige and Gosset numbered forty-three. Déjerine and Mouzon[6] did not cite the number of cases observed, but merely stated their findings in various nerves. Neither recorded the individual cases.

The patients in this series had been injured at least six months prior to examination so that degeneration was completed long before they presented themselves. Most of the cases had been at a standstill for a considerable period, and were therefore deemed ready for operative interference. One case (Case 49) was in a stage of partial regeneration after a previous suture.

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[5] Kraus, W. M., and Ingham, S. D.: Electrical Stimulation of Peripheral Nerves Exposed at Operation: "Surgical Value," J. A. M. A. **74**:586 (Feb. 28) 1920.

[6] Déjerine, M. and Mme., and Mouzon: Les lesions des gros troncs nerveux des members par projectiles de guerre, Presse Méd., May 10, July 8 and August 30, 1915.

TABLE 1.—Number of Reactions in Nerves and Muscles in Fifty-Eight Operative Cases

	Case Numbers	Nerve	Muscles and Sensations	Total
I. Brachial plexus	5, 41	5		
1. Pectoralis major		..	1	
			—	1
II. Median nerve:				
(a) Outer head	31	1		
(b) Inner head	11, 31	2		
(c) Trunk	2, 3, 9, 13, 16, 17, 19, 26, 27, 30, 36, 43, 44, 45, 50, 53, 67, 69	18		
1. Pronator radii teres		..	14	
2. Flexor carpi radialis		..	12	
3. Palmaris longus		..	3	
4. Flexors of the fingers		..	11	
5. Flexor longus pollicis		..	7	
6. Pronator quadratus		..	2	
7. Abductor pollicis		..	2	
8. Opponens pollicis		..	1	
9. Lumbricales		..	1	
10. Sensory		..	3	
			—	56
III. Ulnar nerve	3, 8, 11, 13, 16, 18, 27, 28, 31, 34, 35, 42, 43, 45, 47, 49, 50, 60, 66	19		
1. Flexor carpi ulnaris		..	15	
2. Flex. profundus digitorum		..	13	
3. Adductor pollicis		..	6	
4. Interossei		..	5	
5. Hypothenars		..	4	
6. Sensory		..	1	
			—	44
IV. Circumflex nerve	5, 31, 39	3		
1. Deltoid		..	3	
			—	3
V. Musculospiral nerve	15, 22, 31, 33	4		
1. Triceps		..	4	
2. Supinator longus		..	2	
3. Extensor carpi radialis		..	2	
4. Extensors of the fingers		..	1	
			—	9
VI. Sciatic nerve:				
(a) Internal popliteal divis.	12, 20, 23, 24, 64	5		
1. Hamstrings		..	3	
2. Gastrocnemius		..	4	
3. Tibialis posticus		..	3	
4. Flexor longus digit.		..	2	
5. Flex. longus hallucis		..	2	
6. Intrinsics		..	0	
			—	14
(b) Ext. popliteal division.	14, 21, 59	3		
1. Peronei		..	3	
2. Tibialis anticus		..	2	
3. Extensor digitorum.		..	1	
4. Extensor hallucis		..	1	
			—	7
VII. Internal popliteal nerve	1, 6, 7, 10, 40, 51, 52, 54, 56, 58, 63, 64, 68	12		
1. Hamstrings		..	1	
2. Gastrocnemius and soleus		..	9	
3. Tibialis posticus		..	10	
4. Flexor longus digitorus		..	5	
5. Flexor longus hallucis		..	4	
6. Intrinsics		..	2	
			—	31
VIII. External popliteal nerve	10, 14, 38, 46, 59	5		
1. Peronei		..	5	
2. Tibialis anticus		..	2	
3. Extensor digitorum		..	1	
4. Extensor hallucis		..	1	
			—	9
Total		77	..	174

LEVELS OF STIMULATION

The key to the level of stimulation of the different nerves may be found in the diagrams (Figs. 2 and 3). It will be seen that the various nerves have been stimulated at practically *all levels.* The case reports are arranged in order of the level of stimulation beginning at the proximal part of the nerve. The stimulation was applied to the nerve trunks a little below and a little above the lesions, as well as on the injured portions of the nerves.

ELECTRIC PHENOMENA

The electric phenomena which were brought to light by this work have been briefly commented on in our previous paper.[5] These phenomena were observed:

1. A phenomenon of difference between direct and percutaneous stimulation, fourteen cases (2, 3, 5, 8, 9, 15, 21, 24, 26, 31, 36, 39, 43, 49). The importance of this phenomenon when operating on nerves is clear. When a fasciculus, which is inactive on stimulation through the skin, is found to be active on stimulating the nerve directly, its conductivity is proven and should not be cut. The converse, that absence of any reaction indicates no live fibers, is not necessarily true for obvious reasons. This phenomenon must also be interpreted in each case in terms of the following one.

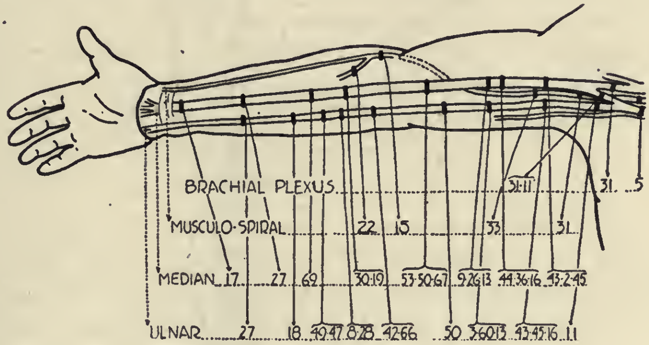

Fig. 2.—Level of stimulation of the principle nerves of the arm and their branches.

2. A direct Erb's paradoxical phenomenon. In this condition some muscles reacted when the nerve was directly stimulated *below* the lesion, which did not react when the nerve was directly stimulated *above* the lesion. Thus the lesion acted as a delaying agent for the current (Cases 13 and 28).

A discussion of the significance of these two phenomena for nerve physiology would consume too much space. Their practical importance requires no discussion.

PLEXUS FORMATIONS IN NERVES, FASCICULI AND FIBERS

The question of internal nerve plexuses is a crucial one in the problem of fascicular anatomy and physiology.

In monosegmental mixed nerves, the deep branches (to muscles) contain, besides motor fibers, those carrying proprioceptive sensations and pain. Since these sensory fibers are of posterior root origin while the motor fibers are of anterior root origin, it is obvious that there must be an *internal nerve plexus* due to the change from the condition of root fasciculi at that of nerve fasciculi. Sensory fibers must pass to motor fasciculi. This demands one type of internal nerve plexus. A second type of internal nerve plexus must occur on account of the interfascicular rearrangement of different sensory functional groups of fibers as the functional arrangement in the spinal cord changes to that of various nerves.

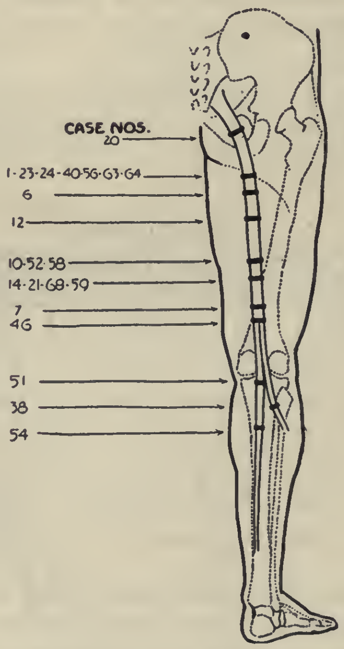

Fig. 3.—Levels of stimulation of the sciatic nerve and its branches.

When polysegmental nerves are brought to our attention, it becomes evident that fibers from two or more segments to one muscle or skin area (through some cutaneous nerve) must reach a fasciculus which goes to this muscle or skin area. This implies a third type of internal nerve plexus.

A more lengthy discussion of the many problems and a review of the literature will be postponed for a later paper.

PREPONDERANCE OF REACTION IN PROXIMAL MUSCLES

As a general rule it has been found that stimulation of the proximal part of the nerve does not cause contraction of muscles supplied by its distal portion. This rule has been found to apply to most nerves examined (see Table 1), and the individual case reports indicate this. Exceptions to the rule have occurred when mild currents were used, but the question of technic is apparently not the only factor since the exceptions were so rare. It seems probable that the contractions of the more powerful proximal muscles mask the actions of the smaller hand and foot muscles. Another factor may be the central localization of their fasciculi. Though we have no anatomic data to support the latter supposition, it seems probable that fasciculi for the most distal parts should in general be most centrally located.

STRAIGHT COURSE OF MOTOR FASCICULI

A review of the results for a given muscle at various levels brings out one point with certainty—*the course is a straight one from the point where the nerve has been made up by its contributing segments to the point of offset of the fasciculi as a branch.* An exception to this was uniformly found in one instance in which it was to be expected, namely, the fasciculi to the pronator radii teres. Further comment will be made in discussing the median nerve.

The straight course of fasciculi was commented on briefly by Marie in the case of the sciatic nerve. Stoffel's anatomic dissections implied it, but that is all. As far as we know, this point has not received the emphasis it deserves when its great importance for the knowledge of peripheral nerve anatomy is considered.

The reader may observe the basis of this statement by starting with a picture showing the results at the proximal end of a nerve and tracing the position of some particular bundle to the point of leaving the main trunk. Figures 40, 41, 42, 43, 45, 47, 48, 49, 51, 54, 55, 56 for example, will show the course of the fibers to the flexor carpi ulnaris. Slight variations in position are no doubt due to slight rotation of the nerve trunk.

FINDINGS IN INDIVIDUAL NERVES AND THEIR BRANCHES

The nerves supplying the upper and lower extremities were stimulated at different points, from the vertebral foramina to the individual branches as they approached their muscles. The results of stimulation at the different levels were:

1. *Roots of the Brachial Plexus.*—One case, Case 41 (Figs. 4, 5 and 6).

The fifth and sixth cervical roots were not stimulated successfully.

Stimulation of the seventh cervical root gave contractions of the pectoral and triceps muscles.

Stimulation of the eighth cervical root at first caused contraction of the finger extensors, mainly of the extensor indicis; subsequent stimulations caused flexion of the fingers and thumb.

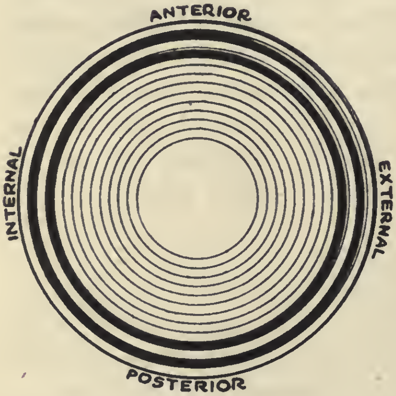

Figure 4

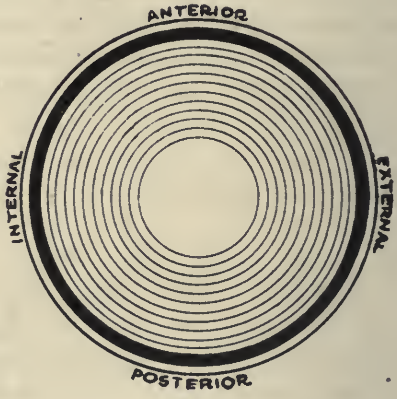

Figure 5

Fig. 4 (Case 41).—Normal brachial plexus, seventh cervical root; pectoralis major and triceps.

Fig. 5 (Case 41).—Normal brachial plexus, eighth cervical root; flexion of fingers and thumb.

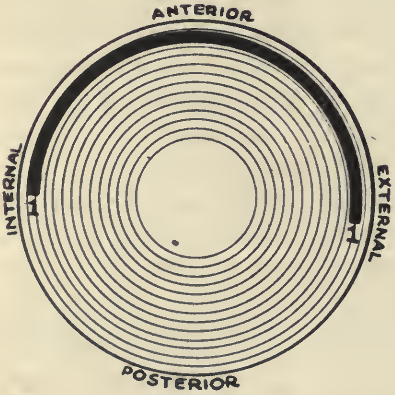

Figure 6

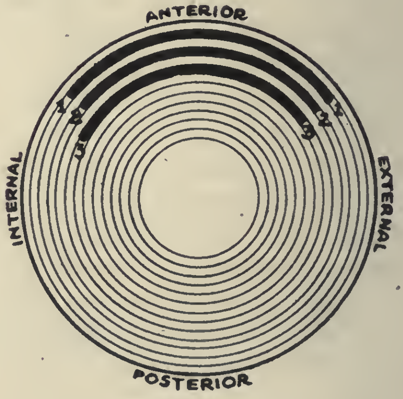

Figure 7

Fig. 6 (Case 41).—Normal brachial plexus, first thoracic root; 1, flexion of fingers and wrist.

Fig. 7. (Case 5).—Abnormal brachial plexus, posterior cord: (1) triceps; (2) deltoid; (3) supinator longus.

The first thoracic root was associated with flexion of the fingers and wrist.

No observations were made on the primary trunks of the brachial plexus.

Cords of the Brachial Plexus: One case; Case 5 (Figs. 7 and 8).

The posterior cord of the brachial plexus was stimulated in one case resulting in contraction of the deltoid, triceps and supinator longus muscles. The lower cord was also stimulated in this case, resulting in flexion of the index and middle finger, on its inner aspect.

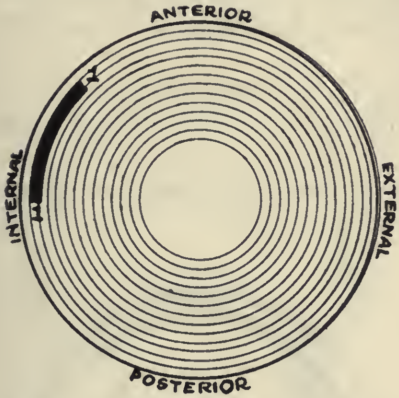

Figure 8

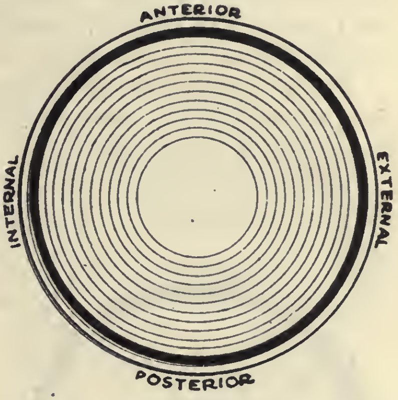

Figure 9

Fig. 8 (Case 5).—Abnormal brachial plexus, lower cord: (1) flexion of index and middle finger.

Fig. 9 (Case 31).—Normal median nerve, external head; pronator radii teres.

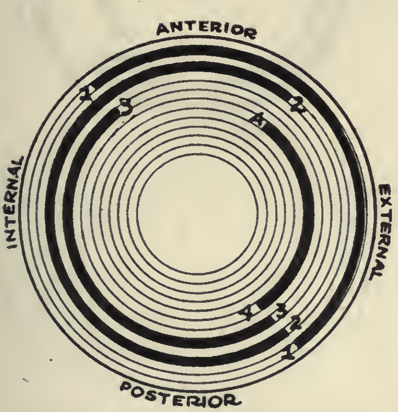

Figure 10

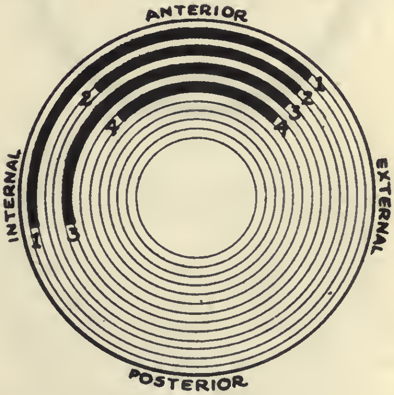

Figure 11

Fig. 10 (Case 31).—Abnormal median nerve, inner head: (1) flexor carpi radialis; (2) flexion of the fingers; (3) flexion of the thumb; (4) abductor pollicis.

Fig. 11 (Case 11).—Abnormal median nerve, inner head: (1) flexor carpi radialis; (2) palmaris longus; (3) flexor longus digitorum; (4) flexor longus pollicis.

Inner and Outer Heads of the Median Nerve: The external head of the median nerve was stimulated in one case, *Case 31* [7] (Fig. 9), resulting in the contraction of the pronator radii teres over its entire circumference. Marie, Meige and Gosset also found this muscle represented in the external head.

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[7] Normal cases are italicized.

The inner head of the median nerve was stimulated twice; in both instances it was abnormal. In one case, Case 31 (Fig. 10), contraction resulted in the flexor carpi radialis, flexors of the fingers, thumb and the abductor pollicis. In another case, Case 11 (Fig. 11), the inner head of the median was abnormal and the result was contraction of the flexor carpi radialis, palmaris longus and flexors of the fingers and thumb. In neither instance was there a reaction in the pronator radii teres. Marie, Meige and Gosset found the flexors of the wrist and fingers in the inner head.

From the results of the stimulation of the roots, the different portions of the brachial plexus and the two heads of the median, certain conclusions may be drawn:

Flexion of the fingers results chiefly from the stimulation of the eighth cervical and first thoracic nerves.

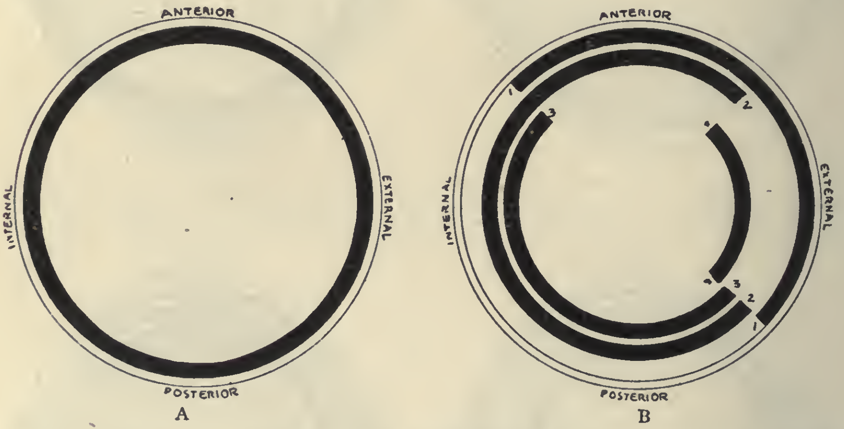

Fig. 12.—Results of stimulation of the two heads of the median nerve: *A*, external (from C6 and 7) pronator radii teres; *B*, internal (C8 and D1); (1) flexor carpi radialis; (2) flexors of the fingers; (3) flexion of the thumb; (4) abductor pollicis.

The triceps and pectoralis major react from the seventh cervical root and not from the eighth cervical and first thoracic. Sherren has observed slight contractions of these muscles on stimulating the sixth cervical root.

In stimulating the posterior cord, contraction occurs in the deltoid, triceps and supinator longus.

The external head of the median evidently supplies only the pronator radii teres muscle, indicating its origin from the sixth and seventh cervical segments (Fig. 12).

The inner head, receiving fasciculi from the eighth cervical and first thoracic roots, supplies all of the long forearm median muscles, in addition to the abductor pollicis, indicating that the supply of the last named muscle as given in textbooks as the sixth and seventh cervical is not entirely correct. It certainly receives fibers from the eighth cervical and first thoracic. *This observation would explain the absence of involvement of the thumb muscles in the Erb type of brachial plexus paralysis.*

2. *Median.*—Eighteen cases; Cases 2 (Fig. 14); *3* (Fig. 20); 9 (Fig. 22), 13 (Fig. 19), 16 (Fig. 16), 17 (Figs. 34, 35 and 36), 19 (Figs. 26, 27 and 28), 26 (Fig. 21), 27 (Fig. 31, 32 and 33), 30 (Fig. 29), 36 (Fig. 17), 43 (Fig. 15), 44 (Fig. 18), 45 (Fig. 13), 50 (Fig. 24), 53 (Fig. 25), 67 (Fig. 23) and 69 (Fig. 30).

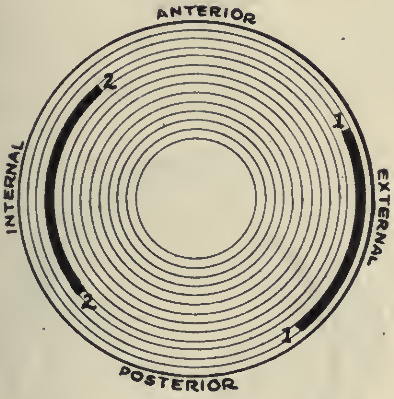

Figure 13

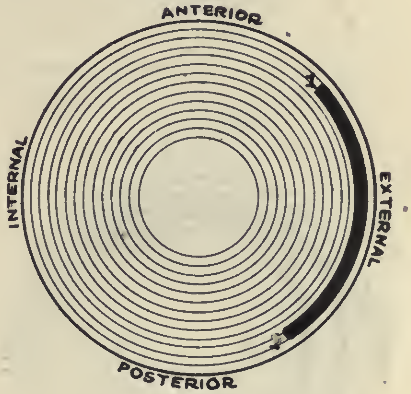

Figure 14

Fig. 13 (Case 45).—Abnormal median nerve: (1) pronator radii teres; (2) flexor profundus digitorum.

Fig. 14 (Case 2).—Abnormal median nerve: (1) pronator radii teres.

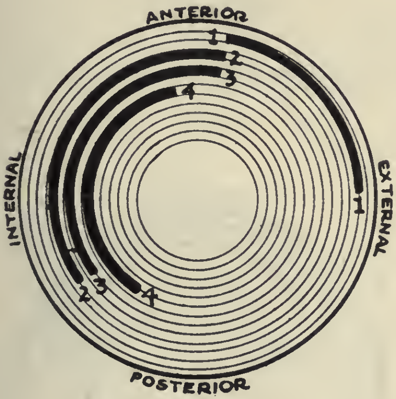

Figure 15

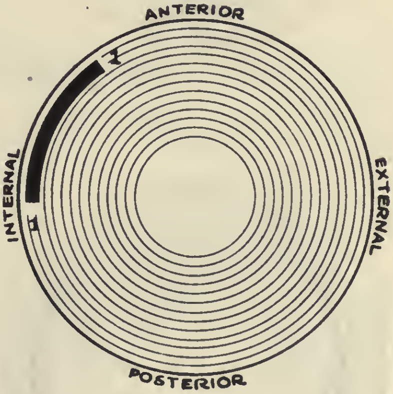

Figure 16

Fig. 15 (Case 43).—Abnormal median nerve: (1) pronator radii teres; (2) flexor carpi radialis; (3) flexion of the fingers; (4) flexor longus pollicis.

Fig. 16 (Case 16).—Abnormal median nerve: (1) pronator radii teres.

(*a*) Pronator Radii Teres: Fourteen cases; Cases 2 (Fig. 4), *3* (Fig. 20), 9 (Fig. 22), 13 (Fig. 19), 26 (Fig. 21), 31 (Fig. 10), 36 (Fig. 17), 43 (Fig. 15), 44 (Fig. 18), 45 (Fig. 13), 50 (Fig. 24), 53 (Fig. 25), 67(Fig. 23).

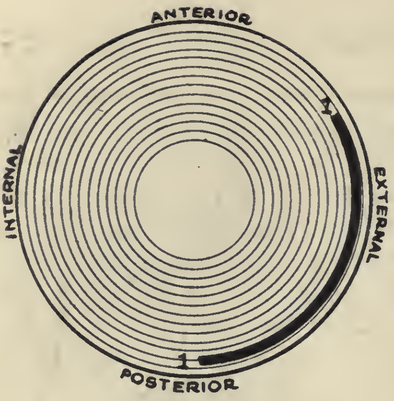

Figure 17

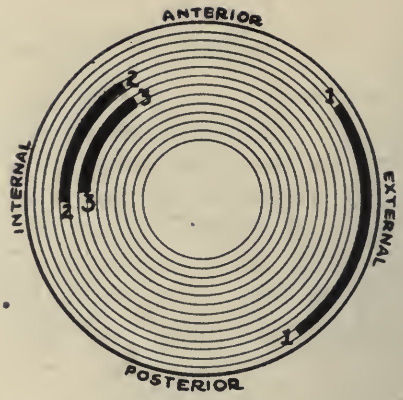

Figure 18

Fig. 17 (Case 36).—Abnormal median nerve: (1) pronator radii teres.

Fig. 18 (Case 44).—Abnormal median nerve: (1) pronator radii teres; (2) flexor sublimis digitorum; (3) flexor carpi radialis.

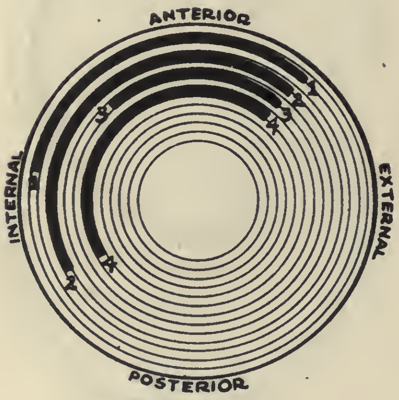

Figure 19

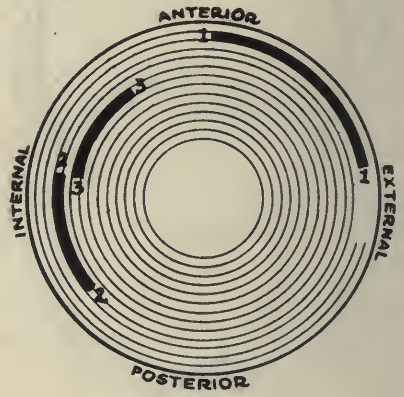

Figure 20

Fig. 19 (Case 13).—Abnormal median nerve: (1) pronator radii teres (greatest reaction antero-internally); (2) flexor carpi radialis; (3) palmaris longus; (4) flexion of the fingers.

Fig. 20 (Case 3).—Normal median nerve: (1) pronator radii teres; (2) flexor carpi radialis; (3) flexor longus pollicis.

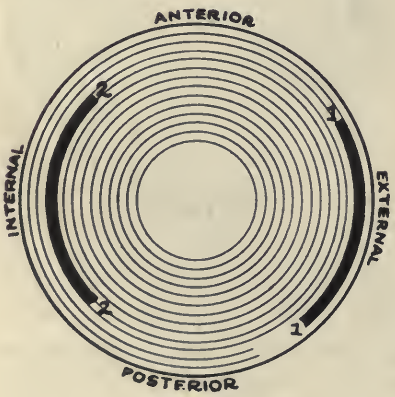

Figure 21

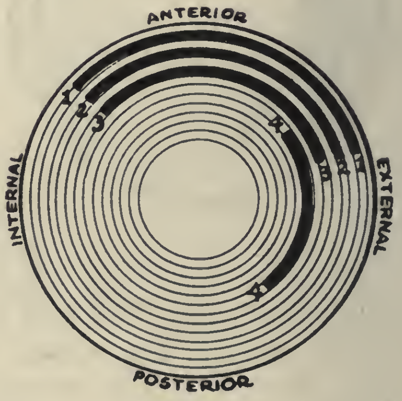

Figure 22

Fig. 21 (Case 26).—Abnormal median nerve: (1) pronator radii teres (above lesion); (2) pronator radii teres (below lesion).

Fig. 22 (Case 9).—Abnormal median nerve: (1) flexion of wrist; (2) flexion of fingers; (3) flexion of thumb; (4) pronator radii teres.

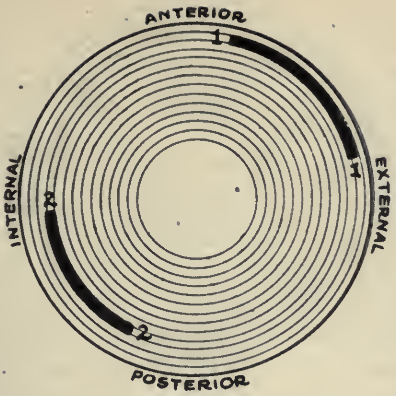

Figure 23

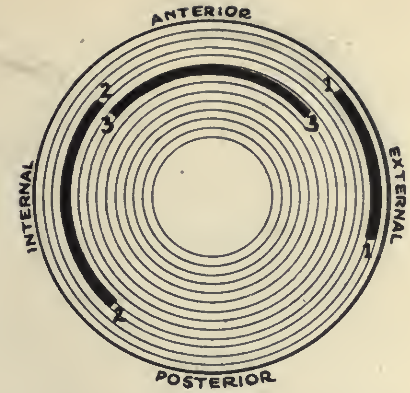

Figure 24

Fig. 23 (Case 67).—Abnormal median nerve: (1) pronator radii teres; (2) flexor carpi radialis.

Fig. 24 (Case 50).—Abnormal median nerve: (1) pronator radii teres; (2) flexor carpi radialis; (3) flexor profundus digitorum.

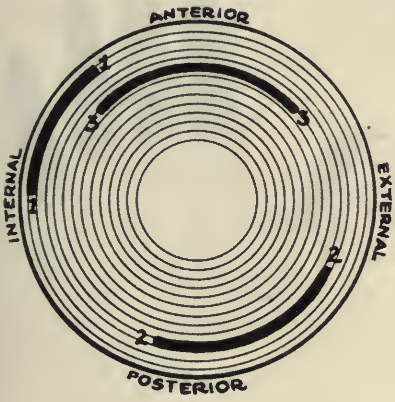

Figure 25

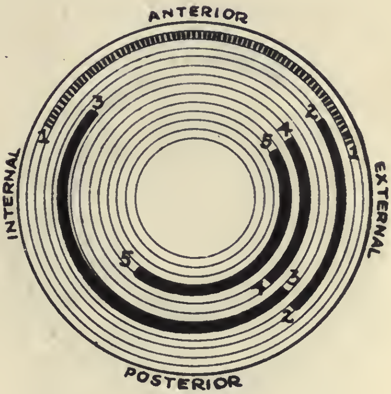

Figure 26

Fig. 25 (Case 53).—Abnormal median nerve: (1) pronator radii teres; (2) flexor carpi radialis; (3) flexor profundus digitorum.

Fig. 26 (Case 19).—Abnormal median nerve: (1) sensory; (2) pronator quadratus; (3) opponens pollicis; (4) abductor pollicis; (5) lumbricales 1 and 2.

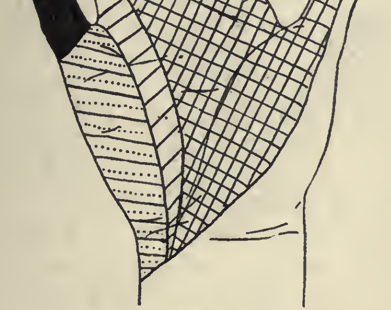

Figure 27

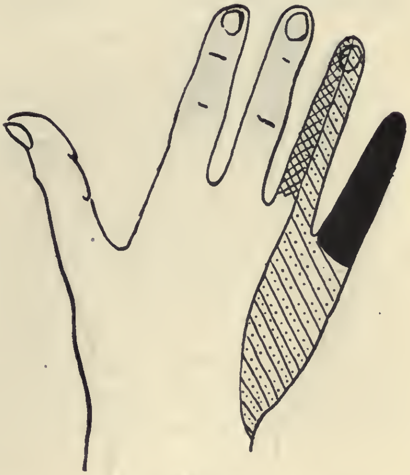

Figure 28

Fig. 27 (Case 19).—Black part denotes complete anesthesia; lines denote brush anesthesia; dots, pin-prick anesthesia (15 gm. of pressure).

Fig. 28 (Case 19).—Crossed lines denote hyperesthesia; black part, complete anesthesia; lines, brush anesthesia; dots, pin-prick anesthesia (15 gm. pressure).

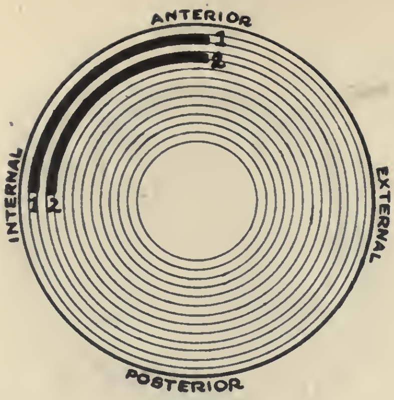

Figure 29

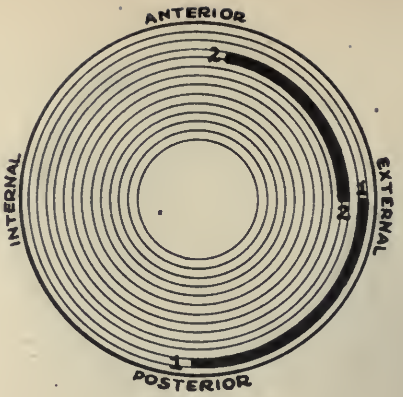

Figure 30

Fig. 29 (Case 30).—Abnormal median nerve: (1) flexor carpi radialis; (2) palmaris longus.

Fig. 30 (Case 69).—Abnormal median nerve: (1) pronator quadratus; (2) flexor longus pollicis.

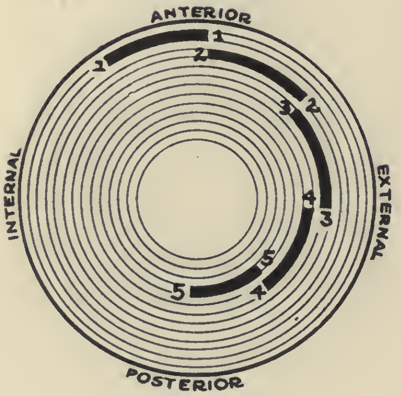

Figure 31

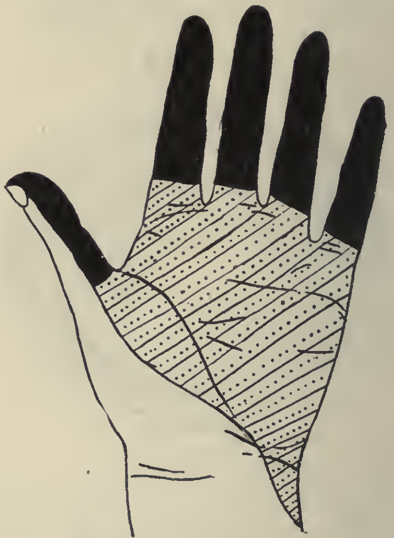

Figure 32

Fig. 31 (Case 27).—Abnormal median nerve; sensory findings—pain.

Fig. 32 (Case 27).—Black part denotes complete anesthesia; lines, brush anesthesia, dots, pin-prick anesthesia (15 gm. pressure).

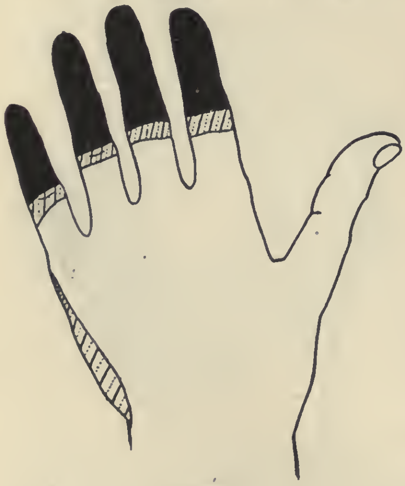

Figure 33

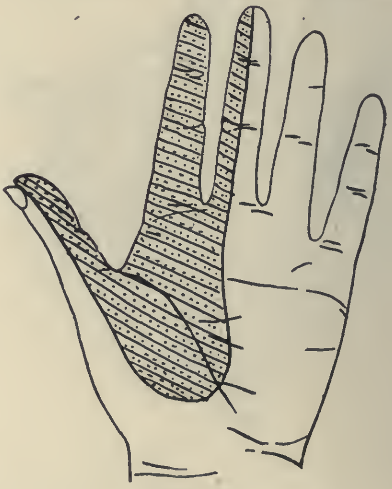

Figure 34

Fig. 33 (Case 27).—Black part denotes complete anesthesia; lines, brush anesthesia, dots, pin-prick anesthesia (15 gm. pressure).

Fig. 34 (Case 17).—Lines denote brush anesthesia; dots, pin-prick anesthesia (15 gm. pressure).

This muscle has been stimulated from the external head of the median to the point at which the branch goes to the muscle. The branches come from the external head of the median (cervical 6 and 7) and leave the nerve internally. They must therefore cross the nerve as they descend. One of these cases, stimulated above the upper third arm, showed this fasciculus on the external aspect. From here to the elbow, two cases show a response on the anterior surface of the nerve. In one case (Case 26, Fig. 21) the muscle responded both externally and internally, the former when stimulated rather high above the lesion, the latter when stimulated low, below the lesion. Anatomic dissection has borne out these observations, and the fibers are found passing from the external to internal part on the anterior surface of the nerve. Marie, Meige and Gosset found both pronators antero-external. Stoffel found the pronator radii teres on the external surface. He has shown the crossing which we have confirmed, in one of the figures in his article.

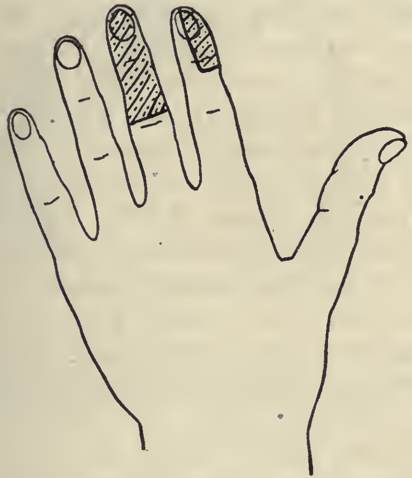

Figure 35

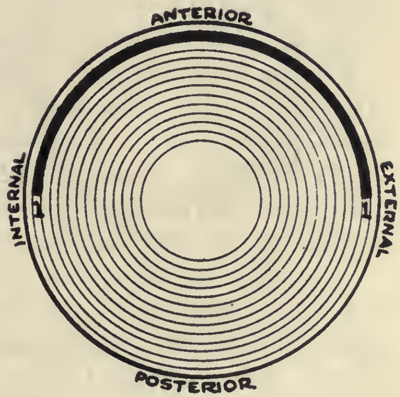

Figure 36

Fig. 35 (Case 17).—Lines denote brush anesthesia; dots, pin-prick anesthesia (15 gm. pressure).

Fig. 36 (Case 17).—Abnormal median nerve: (1) sensory findings—pain.

(*b*) Flexor Carpi Radialis: Twelve cases; Cases *3* (Fig. 20), 9 (Fig. 22), 11 (Fig. 1), 13 (Fig. 19), 16 (Fig. 16), 30 (Fig. 29, 31 Fig. 10), 43 (Fig. 15), 44 (Fig. 18), 50 (Fig. 24), 53 (Fig. 25), 67 (Fig. 23).

The fibers supplying this muscle have been found occupying an antero-internal position in the trunk of the median nerve. Stoffel places them externally, and Marie, Meige and Gosset are uncertain whether they are internal or external.

(*c*) Flexor Sublimis and Flexor Profundus Digitorum: Eleven cases: Cases 5 (Fig. 8), 9 (Fig. 22), 11 (Fig. 11), 13 (Fig. 19), 31 (Fig. 10), 41 (Figs. 5 and 6), 43 (Fig. 15), 44 (Fig. 18), 45 (Fig. 13), 50 (Fig. 24), 53 (Fig. 25).

These muscles have been considered together as it is impossible to distinguish the difference in their action under the conditions existing during a surgical operation. The position of fibers to these muscles has been found to be antero-internal. Marie, Meige and Gosset found these postero-internally. Stoffel puts the profundus postero-internally, but the sublimis internally. Two cases were stimulated through the brachial plexus. (Case 41, Figs. 5 and 6; Case 5, Fig. 8).

(*d*) Flexor Longus Pollicis: Seven cases; Case *3* (Fig. 20), 9 (Fig. 22), 11 (Fig. 11), 31 (Fig. 10), 41 (Fig. 6), 43 (Fig. 15), 69 (Fig. 30), These fibers have been found anterior to antero-internal. Stoffel found these fibers to be postero-internal. One case was stimulated through the brachial plexus, Case 41 (Fig. 5).

(*e*) Palmaris Longus: Three cases; Cases 11 (Fig. 11), 13 (Fig. 19), 30 (Fig. 29). This muscle has been successfully stimulated from the anterior surface of the median. Stoffel found it to be external.

(*f*) Pronator Quadratus: Two cases; Cases 19 (Fig. 26), 69 (Fig. 30). This muscle was stimulated from the external and postero-external aspects. Stoffel places this in a postero-internal position.

(*g*) Abductor Pollicis: Two cases; Cases 19 (Fig. 26), 31 (Fig. 10). This muscle was stimulated, in Case 19, from the external aspect of the nerve and also from the inner head of the median nerve, external aspect, in Case 31.

(*h*) Opponens Pollicis: One case, Case 19, (Fig. 26). This muscle was stimulated from the postero-internal aspect. Stoffel places all the thenar muscles anteriorly, occupying three fifths of the nerve. Marie, Meige and Gosset place them posteriorly.

(*i*) First and Second Lumbricales: One case, Case 19 (Fig. 26). These muscles were stimulated from the posterior-external aspect of the nerve.

In view of the fact that the action of the lumbricales muscles has been the subject of some discussion, it may be stated that the result of their stimulation in this case, *was flexion of the metacarpophalangeal joints of the first and second fingers.*

Déjerine and Mouzon have made no observations on the nerve. Their findings on other nerves will be quoted hereafter.

Sensory: Three cases, Cases 17 (Fig. 36), 19 (Fig. 26), 27 (Fig. 31). Three patients were operated on under local anesthesia. In two cases, sensory reactions of pain were caused from stimulation of the anterior aspect, the patients stating "it hurts," "it burns." In Case 27, pain was localized at the base of the index, middle, ring and little fingers as the electrode was passed from the posterior aspect around the external to the anterior surface. No reaction of pain resulted from stimulation of the internal surface of the nerve. Stoffel places the sensory bundle anteriorly.

The foregoing observations on the median nerve brings out these points:

1. The bundles of the long forearm muscles lie anteriorly and internally in the trunk of the median nerve (Fig. 37).

2. The bundles of the intrinsic hand muscles are in the main posterior (Fig. 38).

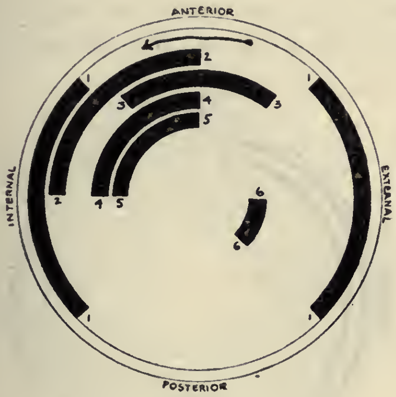

Figure 37

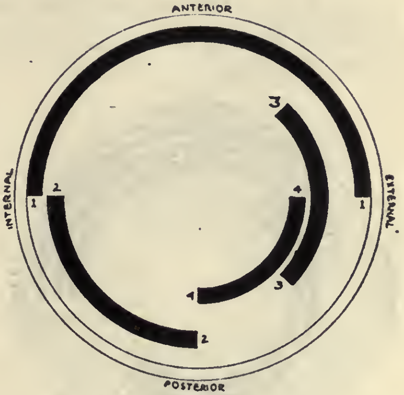

Figure 38

Fig. 37.—Results of stimulation of the median nerve at the midarm level: (1) pronator radii teres; (2) flexor carpi radialis; (3) palmaris longus; (4) flexores digitorum; (5) flexor pollicis; (6) pronator quadratus.

Fig. 38.—Results of stimulation of the median nerve at midforearm level: (1) sensory; (2) opponens pollicis; (3) abductor pollicis; (4) lumbricales.

3. Sensory fibers (pain) are in the main anterior (Fig. 38),

3. *The Ulnar Nerve*: Nineteen cases, Cases 3 (Fig. 46), 8 (Fig. 51), 11 (Fig. 40), 13 (Fig. 44, 16 (Fig. 41), *18* (Fig. 54), 27 (Fig. 57), 28 (Fig. 50), 31 (Fig. 39), 34 (Fig. 55), 35 (Fig. 56), 42 (Fig. 49), 43 (Fig. 43), 45 (Fig. 42), 47 (Fig. 52), 49 (Fig. 53), 50 (Fig. 47), 60 (Fig. 45), 66 (Fig. 48).

This nerve is readily stimulated. Frequently it may be investigated in the normal condition due to its proximity to the median. The results of stimulation have been very satisfactory.

(*a*) Flexor Carpi Ulnaris: Fifteen cases; Cases 3 (Fig. 46), 8 (Fig. 51), 11 (Fig. 40), 16 (Fig. 41), *18* (Fig. 54), 31 (Fig. 39), 34 (Fig. 55), 35 (Fig. 56), 41 (Fig. 6), 42 (Fig. 49), 43 (Fig. 43), 45 (Fig. 42), 50 (Fig. 47), 60 (Fig. 45), 66 (Fig. 48). The position of its fibers is on the internal aspect of the nerve. Marie, Meige and Gosset and Stoffel also find the fibers internal. Déjerine and Mouzon find that they are external.

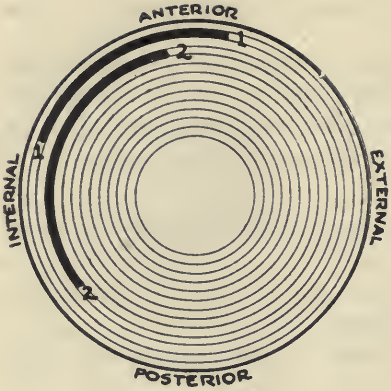

Figure 39

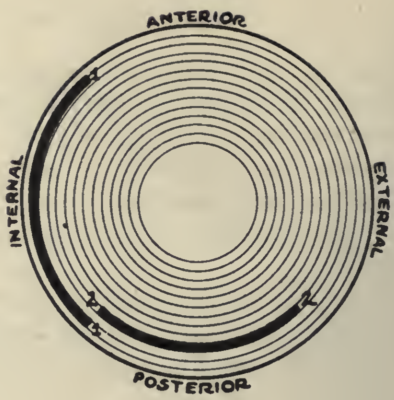

Figure 40

Fig. 39 (Case 31).—Abnormal ulnar nerve: (1) flexor carpi ulnaris; (2) flexor profundus digitorum.

Fig. 40 (Case 11).—Abnormal ulnar nerve: (1) flexor carpi ulnaris; (2) flexor profundus digitorum.

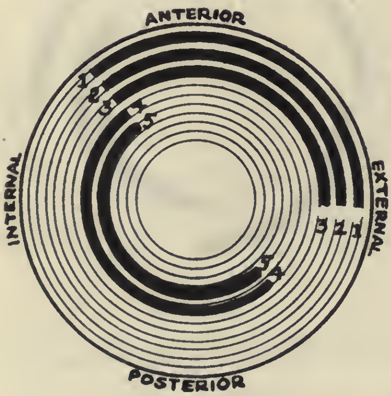

Figure 41

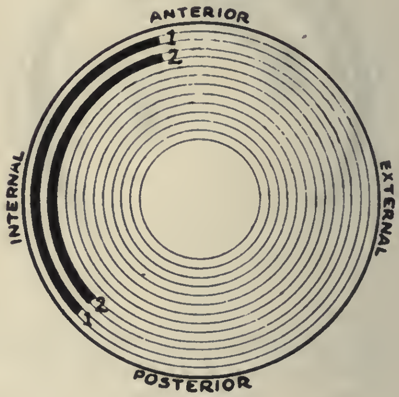

Figure 42

Fig. 41 (Case 16).—Abnormal ulnar nerve: (1) hypothenar muscles; (2) interossei; (3) adductor pollicis; (4) flexor carpi ulnaris; (5) flexor profundus digitorum.

Fig. 42 (Case 45).—Abnormal ulnar nerve: (1) flexor carpi ulnaris; (2) flexor profundus digitorum.

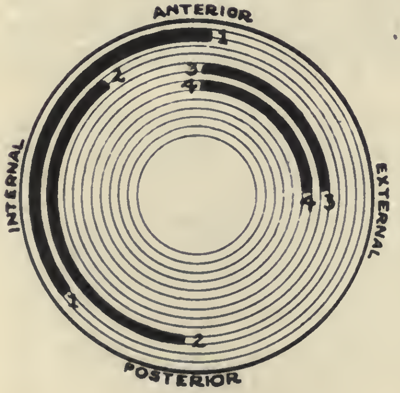

Figure 43

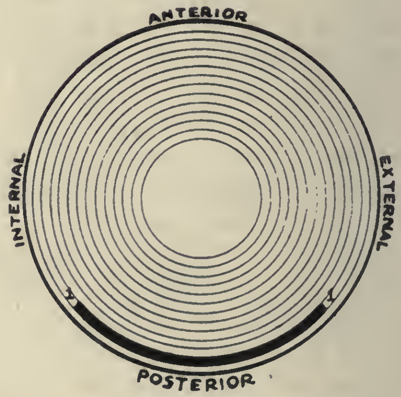

Figure 44

Fig. 43 (Case 43).—Abnormal ulnar nerve: (1) flexor carpi ulnaris; (2) flexor profundus digitorum; (3) adductor pollicis; (4) hypothenar muscles.

Fig. 44 (Case 13).—Abnormal ulnar nerve: (1) flexor profundus digitorum.

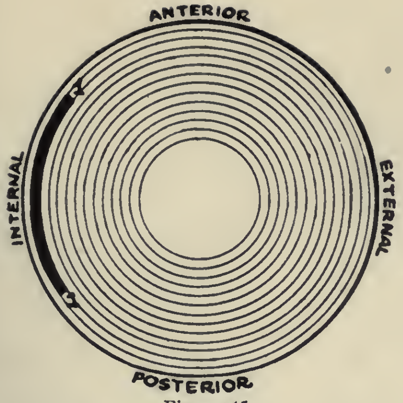

Figure 45

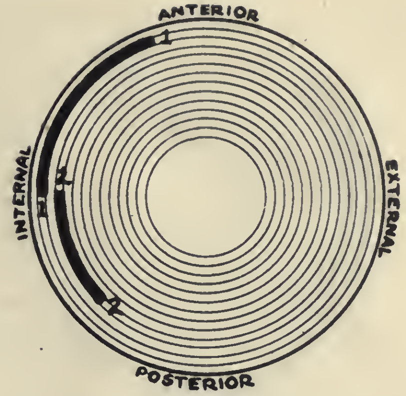

Figure 46

Fig. 45 (Case 60).—Abnormal ulnar nerve: (1) flexor carpi ulnaris.
Fig. 46 (Case 3).—Abnormal ulnar nerve: (1) flexor carpi ulnaris; (2) flexor profundus digitorum.

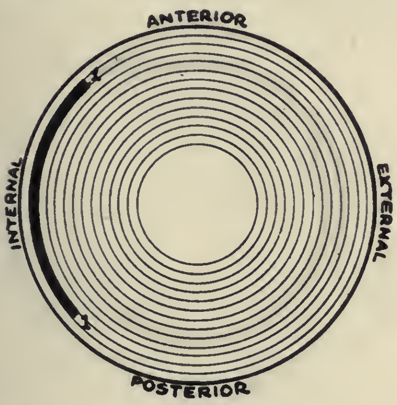

Figure 47

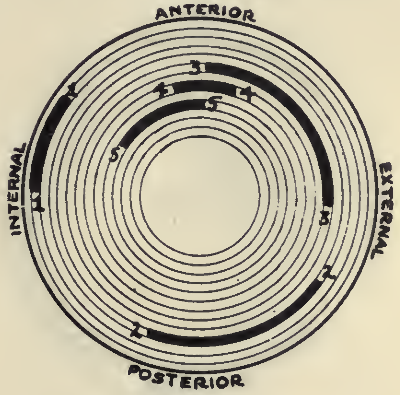

Figure 48

Fig. 47 (Case 50).—Abnormal ulnar nerve: (1) flexor carpi ulnaris.
Fig. 48 (Case 66).—Abnormal ulnar nerve: (1) flexor carpi ulnaris; (2) flexor profundus digitorum; (3) interossei; (4) adductor pollicis; (5) abductor minimi digiti.

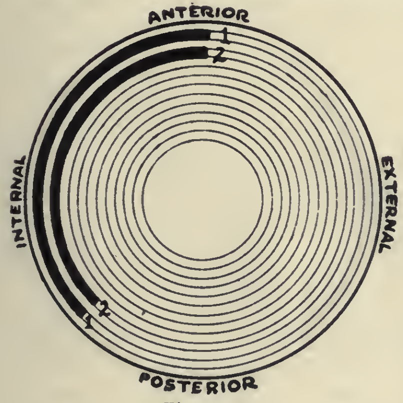

Figure 49

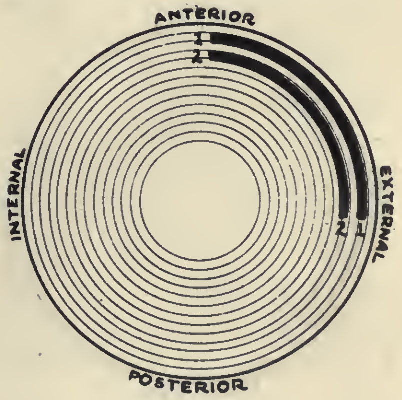

Figure 50

Fig. 49 (Case 42).—Abnormal ulnar nerve: (1) flexor carpi ulnaris; (2) flexor profundus digitorum.
Fig. 50 (Case 28).—Abnormal ulnar nerve: (1) interossei; (2) adductor pollicis.

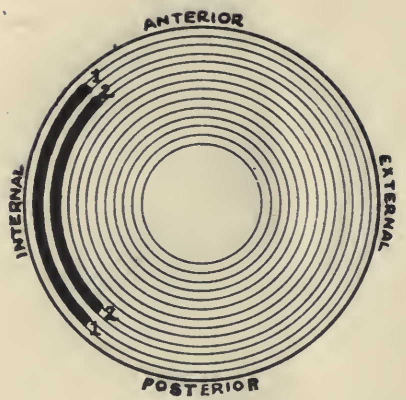

Figure 51

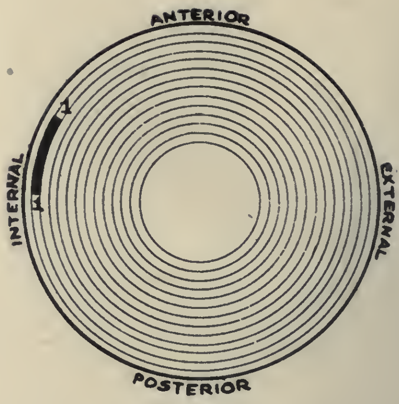

Figure 52

Fig. 51 (Case 8).—Abnormal ulnar nerve: (1) flexor carpi ulnaris; (2) flexor profundus digitorum.

Fig. 52 (Case 47).—Abnormal ulnar nerve: (1) interossei.

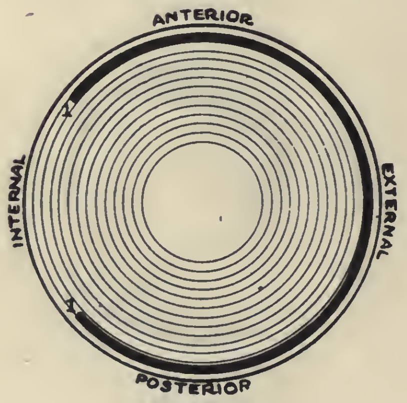

Figure 53

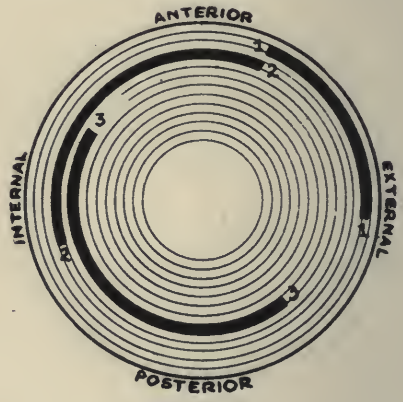

Figure 54

Fig. 53 (Case 49).—Abnormal ulnar nerve: (1) intrinsic muscles of the hand.

Fig. 54 (Case 18).—Normal ulnar nerve: (1) adductor pollicis; (2) flexor carpi ulnaris; (3) flexor profundus digitorum.

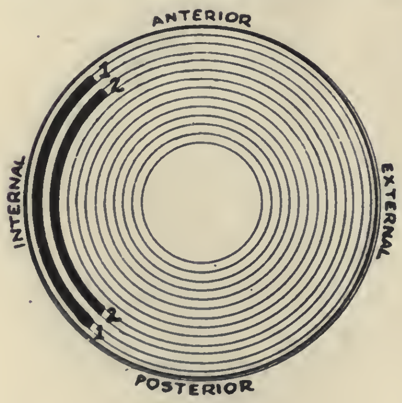

Figure 55

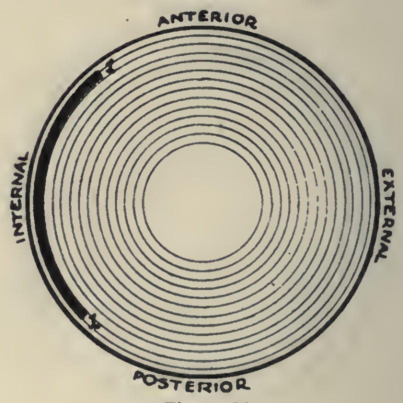

Figure 56

Fig. 55 (Case 34).—Abnormal ulnar nerve: (1) flexor carpi ulnaris; (2) flexor profundus digitorum.

Fig. 56 (Case 35).—Abnormal ulnar nerve: (1) flexor carpi ulnaris.

(*b*) Flexor Profundus Digitorum: Thirteen cases; Cases 3 (Fig. 46), 8 (Fig. 51), 11 (Fig. 40), 13 (Fig. 44), 16 (Fig. 41), *18* (Fig. 54),. 31 (Fig. 39), 34 (Fig. 55), 41 (Fig. 6), 42 (Fig. 49), 43 (Fig. 44), 45 (Fig. 43), 66 (Fig. 48). The fibers of this muscle were found internally, either posterior to those of the flexor carpi ulnaris or in the same position. Marie, Meige and Gosset found that the fibers were postero-external, Déjerine and Mouzon external, and Stoffel internal and posterior to the flexor carpi ulnaris.

(*c*) Adductor Pollicis: Six cases; Cases 16 (Fig. 41), *18* (Fig. 54), 28 (Fig. 50), 43 (Fig. 43), 49 (Fig. 53), 66 (Fig. 48).

The fibers of this muscle lie antero-externally. Déjerine and Mouzon place them postero-externally and Stoffel places them posteriorly.

(*d*) Interossei: Five cases; Cases 16 (Fig. 41), 28 (Fig. 50), 47 (Fig. 52), 49 (Fig. 53) 66 (Fig. 48).

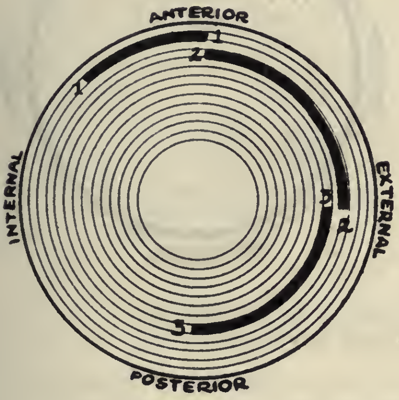

Figure 57

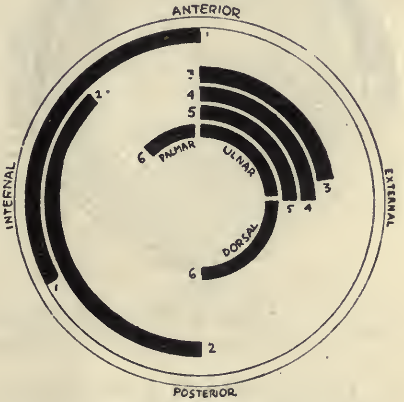

Figure 58

Fig. 57 (Case 27).—Abnormal ulnar nerve, sensory findings in ulnar distribution—pain: (1) palmar surface; (2) ulnar surface; (3) dorsal surface.

Fig. 58.—Results of stimulation of the ulnar nerve: (1) flexor carpi ulnaris; (2) flexor profundus digitorum; (3) hypothenars; (4) interossei; (5) adductor pollicis; (6) sensory.

These fibers are antero-external in all but Case 47, in which these were the only muscles reacting, the rest of the nerve being diseased. This probably accounts for the discrepancy. Déjerine and Mouzon find the fibers for these muscles are internal, Stoffel posterior.

(*e*) Hypothenars: Four cases; Cases 16 (Fig. 41) 43 (Fig. 43), 49 (Fig. 52), 66 (Fig. 48).

These muscles are considered together as it is difficult to obtain sufficiently clear reactions to differentiate their action with certainty. Their fibers are situated antero-externally in the nerve. Déjerine and Mouzon place them internally, Stoffel posteriorly. These muscles were, according to Marie, Meige and Gosset, of uncertain localization but on clinical grounds were placed on the internal and anterior aspect of the nerve.

Sensory: One case; Case 27 (Fig. 57).

Only one case was done under local anesthesia. In this the result was clear cut. On stimulating the nerve antero-internally, pain was felt in the palmar distribution of the ulnar; stimulation antero-externally caused pain on the ulnar aspect of the hand; and postero-externally on the dorsal aspect in the ulnar distribution. As far as we can find, these observations on the location of sensory fibers in the ulnar and median nerves (2) are the first to be recorded. Figure 58 shows the composite results of the ulnar cases.

4. *Circumflex Nerve.*—Three cases; Cases 5 (Fig. 7), *31* (Fig. 59), 39 (Fig. 60).

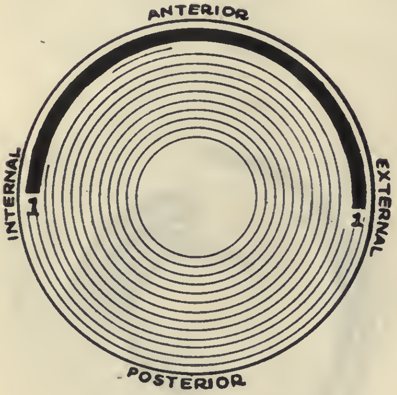

Figure 59

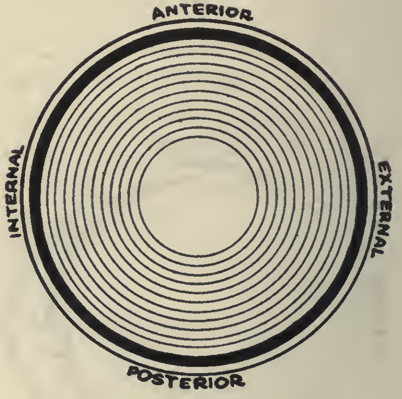

Figure 60

Fig. 59 (Case 31).—Abnormal circumflex nerve: (1) deltoid.
Fig. 60 (Case 39).—Abnormal circumflex nerve: (1) deltoid.

Case 39 showed a reaction in the deltoid on all aspects of the nerve. In Case 31 the deltoid was also stimulated through the brachial plexus. In Case 5 this muscle reacted on the anterior aspect of the posterior cord.

5. *Musculo-Spiral Nerve.*—Four cases; Cases 15 (Fig. 63), 22 (Fig. 64), 31 (Fig. 61), 33 (Fig. 62).

The number of cases of musculo-spiral injury in which a reaction could be obtained was small, the nerve being divided anatomically or physiologically in most instances. Four cases yielded results.

(*a*) Triceps: Four cases; Cases 5 (Fig. 7), 31 (Fig. 61), 33 (Fig. 62), 41 (Fig. 4).

The position of this muscle is easily determined. It is internal, running somewhat posteriorly. The bundle is readily seen. The triceps were also stimulated through the brachial plexus in two cases, Cases 5 (Fig. 7), 41 (Fig. 4).

(*b*) Supinator Longus: Two cases; Case 5 (Fig. 7), Case 31 (Fig. 61).

This muscle reacted in one case and its fibers lay externally. Marie, Meige and Gosset, Déjerine and Mouzon and Stoffel all agree with us. Case 5 was stimulated through the brachial plexus.

(c) EXTENSOR CARPI RADIALIS: Two cases, Cases 31 (Fig. 61), 22 (Fig. 64).

These muscles were found anteriorly in Case 31, posteriorly in Case 22. The discrepancy is probably due to torsion of the nerve as it passes around the humerus. The level of Case 31 was in the axilla, that of Case 22, below the elbow. Marie, Meige and Gosset place these fibers internally; Déjerine and Mouzon and Stoffel postero-externally.

(*d*) EXTENSORS OF THE FINGERS AND THUMB: One case, Case 15 (Fig. 63).

In this case, the extensor indicis reacted on the internal side of the nerve. Marie, Meige and Gosset, Déjerine and Mouzon, and Stoffel found these fibers to be postero-internal.

6. *Sciatic Nerve.*—Twenty-two cases; Cases 1 (Fig. 75), 6 (Fig. 79), *7* (Fig. 85), 10 (Figs. 80, 81, 89), 12 (Fig. 70), 14 (Figs. 71, 90), *20* (Fig. 66), 21 (Fig. 72), 23 (Fig. 67), 24 (Fig. 68), 38 (Fig. 92), *40* (Fig. 76), 46 (Fig. 91), 51 (Fig. 86), 52 (Fig. 82), 54 (Fig. 87), *56* (Fig. 77), 58 (Fig. 83), 59 (Figs. 73, 93), 63 (Fig. 78), *64* Fig. 69), 68 (Fig. 84).

This nerve was stimulated at various levels.

SCIATIC TRUNK: Eight cases; Cases 12 (Fig. 70), 14 (Fig. 71), *20* (Fig. 66), 21 (Fig. 72), 23 (Fig. 69), 24 (Fig. 68), 59 (Fig. 73), *64* (Fig. 69).

1. *Internal Popliteal Division.*[8]— Five cases; Cases 12 (Fig. 70), *20* (Fig. 66), 23 (Fig. 67), 24 (Fig. 68), *64* (Fig. 69).

(*a*) HAMSTRING MUSCLES: Three cases; Cases *20* (Fig. 66), 23 (Fig. 67), *64* (Fig. 69).

The fibers lie postero-internally.

(*b*) GASTROCNEMIUS AND SOLEUS CROUP: Four cases; Cases 12 (Fig. 70), *20* (Fig. 66), 24 (Fig. 68), *64* (Fig. 69).

The fibers were uniformly found on the posterior side internal to the midline between the internal and external division of the nerve.

(*c*) TIBIALIS POSTICUS: Three cases; Cases 12 (Fig. 70), *20* (Fig. 66), *64* (Fig. 69).

In all three cases fibers to this muscle were found anteriorly and internal to the midline of the trunk.

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[8] The use of the word "division" indicates that the two nerves had not been separated and that the trunk was stimulated as a whole.

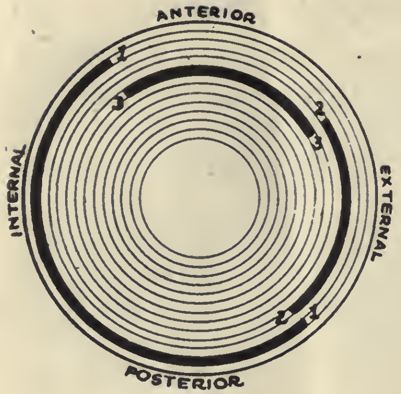


Figure 61

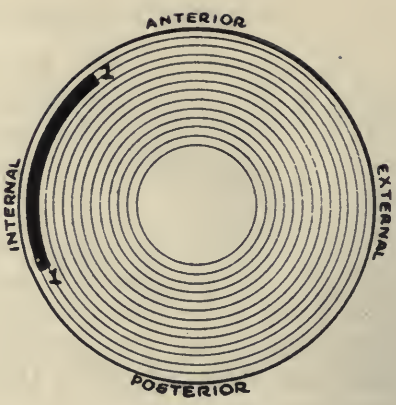


Figure 62

Fig. 61 (Case 31).—Abnormal musculospiral nerve: (1) triceps; (2) supinator longus; (3) extensor carpi radialis.

Fig. 62 (Case 33).—Abnormal musculospiral nerve: (1) triceps.

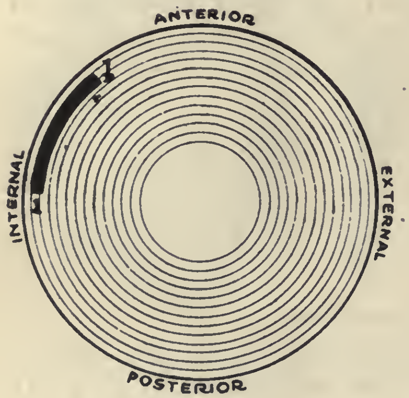


Figure 63

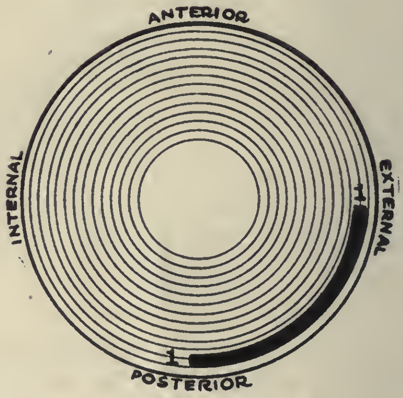


Figure 64

Fig. 63 (Case 15).—Abnormal musculospiral nerve: (1) extensor indicis.

Fig. 64 (Case 22).—Abnormal musculospiral nerve: (1) extensor carpi radialis; greatest reaction posteriorly.

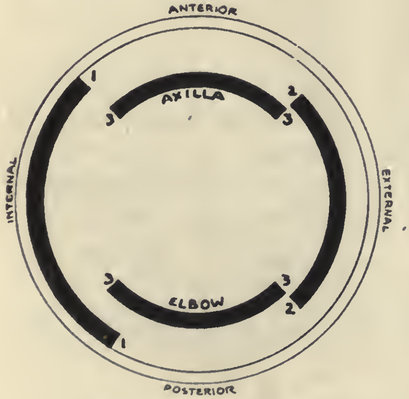


Figure 65

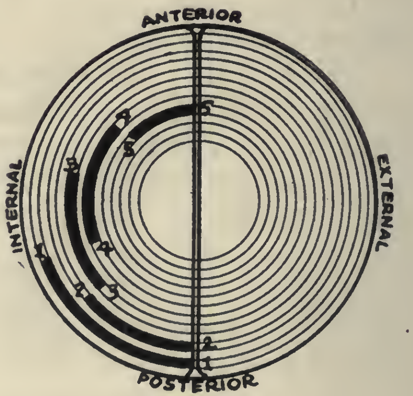


Figure 66

Fig. 65.—Results of stimulation of the musculospiral nerve: (1) triceps; (2) supinator longus; (3) extensor carpi radialis.

Fig. 66 (Case 20).—Normal sciatic nerve: (1) ham strings; (2) gastrocnemius; (3) flexor longus hallucis; (4) flexor longus digitorum; (5) tibialis posticus.

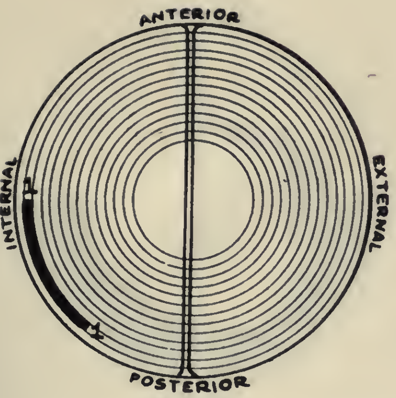


Figure 67

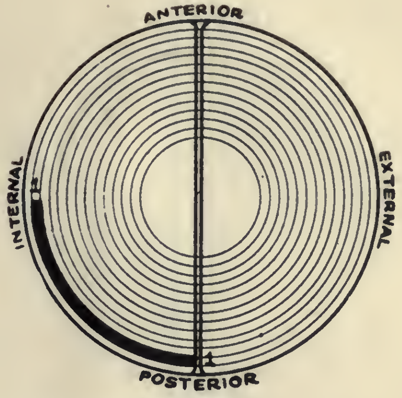


Figure 68

Fig. 67 (Case 23).—Abnormal sciatic nerve: (1) hamstrings.
Fig. 68 (Case 24).—Abnormal sciatic nerve: (1) gastrocnemius and soleus.

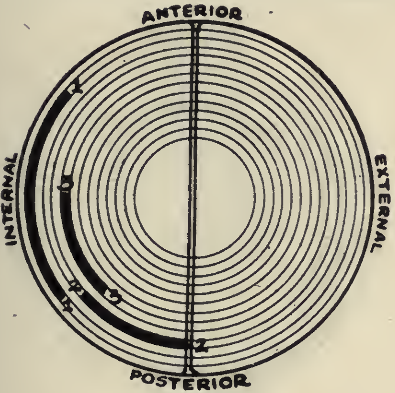


Figure 69

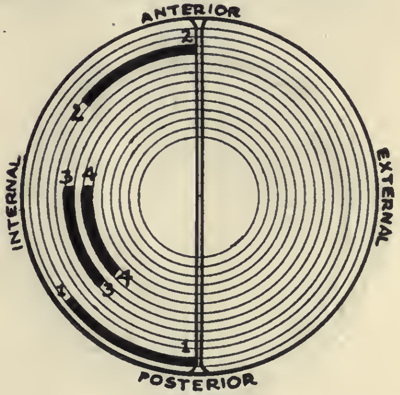


Figure 70

Fig. 69 (Case 64).—Abnormal sciatic nerve: (1) biceps; (2) gastrocnemius; (3) tibialis posticus.

Fig. 70 (Case 12).—Abnormal sciatic nerve: (1) gastrocnemius; (2) tibialis posticus; (3) flexor longus digitorum; (4) flexor longus hallucis.

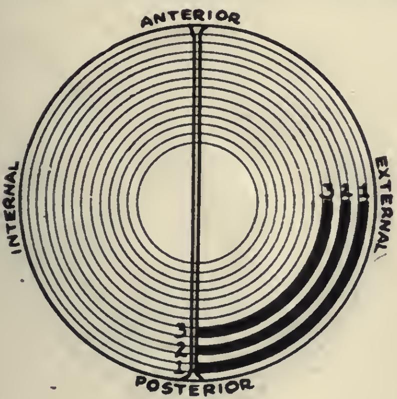


Figure 71

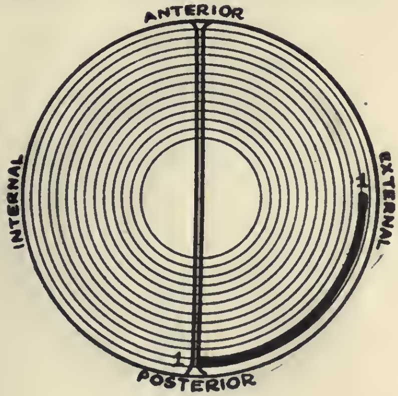
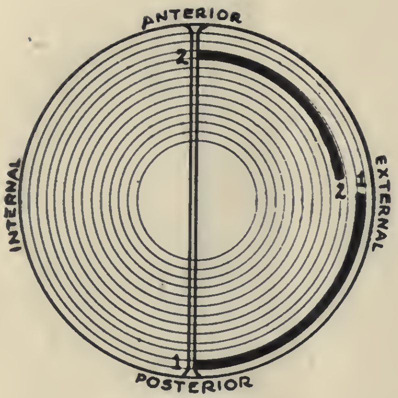


Figure 72

Fig. 71 (Case 14).—Abnormal sciatic nerve: (1) peronei; (2) tibialis anticus; (3) both extensors of the toes.

Fig. 72 (Case 21).—Abnormal sciatic nerve: (1) peronei.



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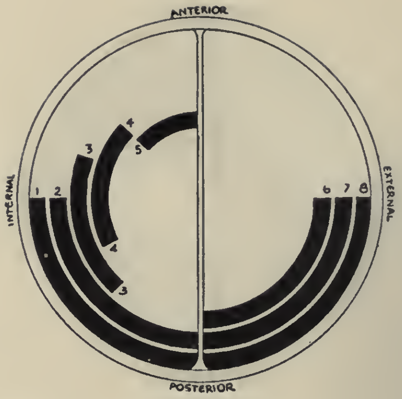
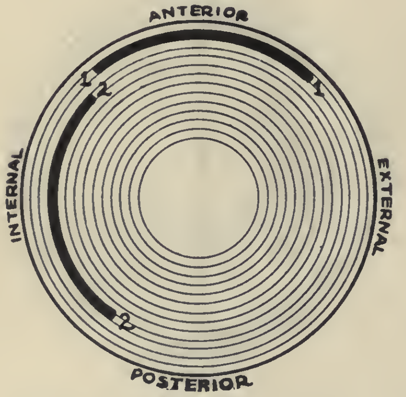


Figure 74

Fig. 73 (Case 59).—Abnormal sciatic nerve: (1) peronei; (2) tibialis anticus.

Fig. 74.—Results of stimulation of the sciatic nerve: (1) hamstrings; (2) gastrocnemius and soleus; (3) flexor longus hallucis; (4) flexor longus digitorum; (5) tibialis posticus; (6) peronei; (7) tibialis anticus; (8) extensors of the toes.



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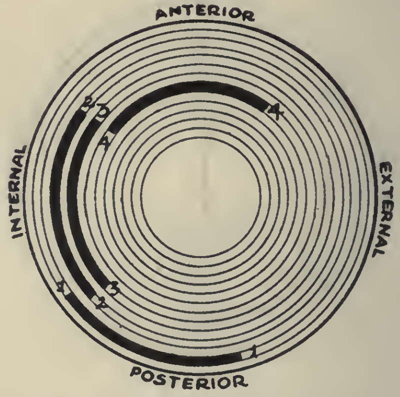


Figure 76

Fig. 75 (Case 1).—Abnormal internal popliteal nerve: (1) tibialis posticus; (2) gastrocnemius and soleus.

Fig. 76 (Case 40).—Normal internal popliteal nerve: (1) gastrocnemius and soleus; (2) tibialis posticus; (3) flexor longus digitorum; (4) flexor longus hallucis.

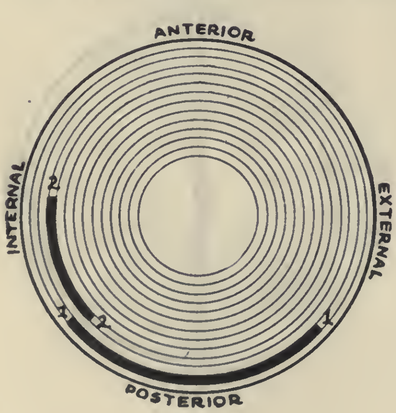


Figure 77

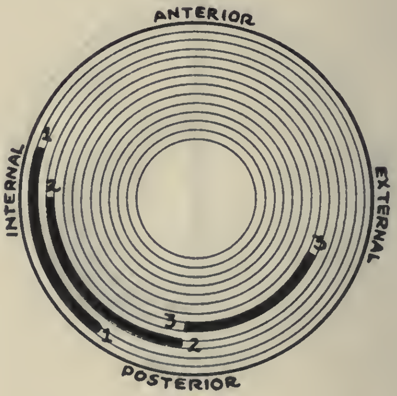


Figure 78

Fig. 77 (Case 56).—Normal internal popliteal nerve: (1) gastrocnemius and soleus; (2) tibialis posticus.

Fig. 78 (Case 63).—Abnormal internal popliteal nerve: (1) biceps femoris; (2) gastrocnemius; (3) tibialis posticus.

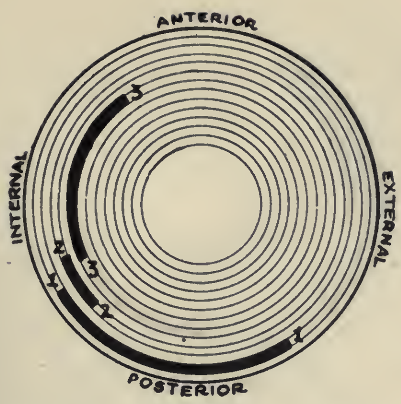


Figure 79

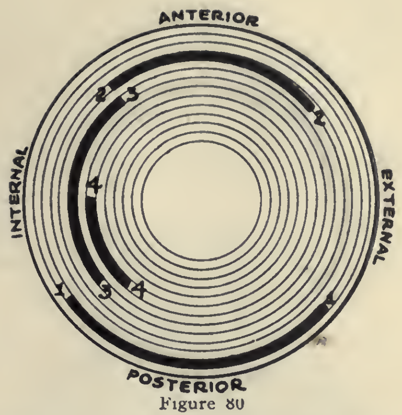


Figure 80

Fig. 79 (Case 6).—Abnormal internal popliteal nerve: (1) tibialis posticus; (2) flexor longus hallucis; (3) flexor longus digitorum.

Fig. 80 (Case 10).—Abnormal internal popliteal nerve: (1) gastrocnemius and soleus; (2) tibialis posticus; (3) flexor longus digitorum; (4) flexor longus hallucis.

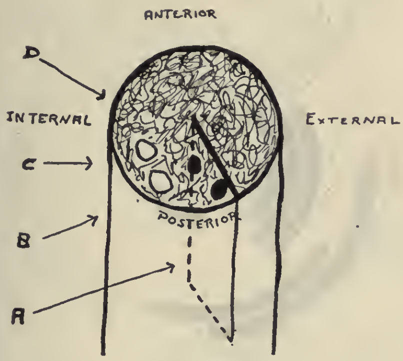


Figure 81

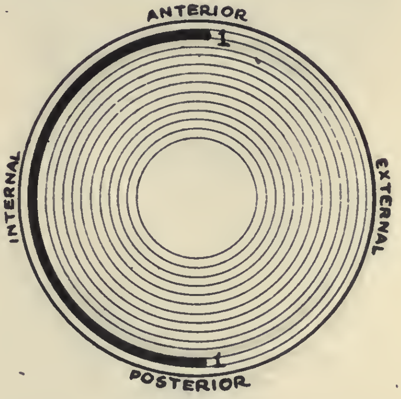
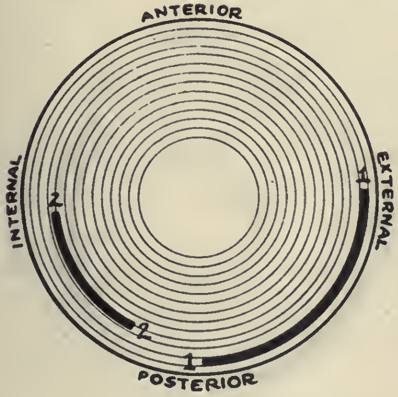


Figure 82

Fig. 81 (Case 10).—*A*, plane of incision in internal popliteal nerve; *B*, the black areas indicate bundles which were normal; *C*, white areas indicate bundles which were abnormal; *D*, interstitial and scar tissue.

Fig. 82 (Case 52).—Abnormal internal popliteal nerve: (1) gastrocnemius and soleus.



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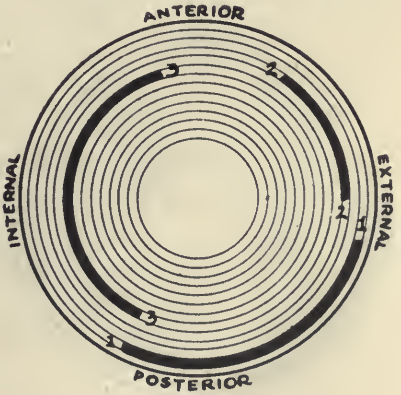

Figure 84

Fig. 83 (Case 58).—Abnormal internal popliteal nerve: (1) Gastrocnemius and soleus; (2) tibialis posticus.

Fig. 84 (Case 68).—Abnormal internal popliteal nerve: (1) gastrocnemius and soleus; (2) tibialis posticus; (3) intrinsic foot muscles.

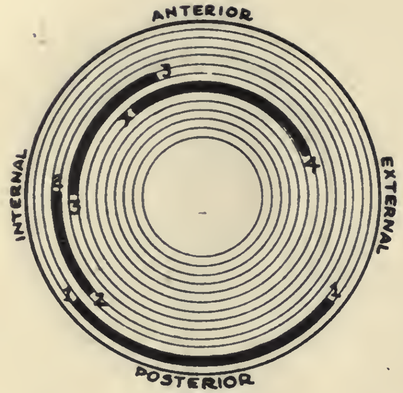

Figure 85

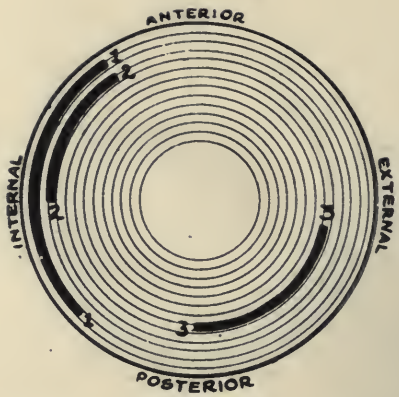

Figure 86

Fig. 85 (Case 7).—Normal internal popliteal nerve: (1) gastrocnemius and soleus; (2) tibialis posticus; (3) flexor longus hallucis; (4) flexor longus digitorum.

Fig. 86 (Case 51).—Abnormal internal popliteal nerve: (1) plantar muscles; (2) flexor minimi digiti; (3) flexor brevis digitorum.

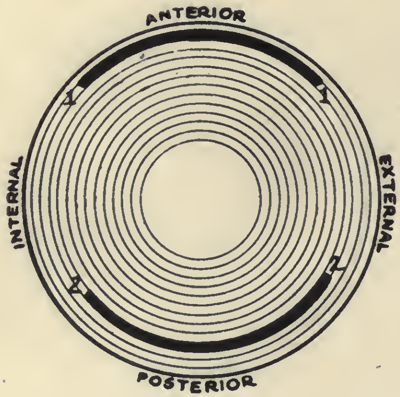

Figure 87

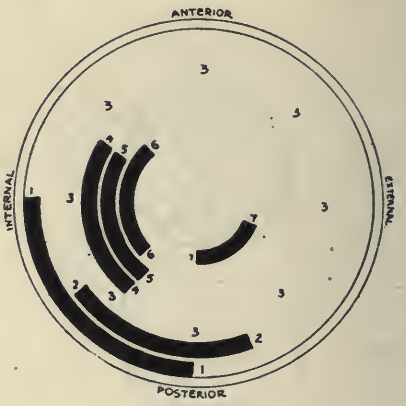

Figure 88

Fig. 87 (Case 54).—Abnormal internal popliteal nerve: (1) tibialis posticus; (2) flexor longus digitorum.

Fig. 88.—Results of stimulation of the internal popliteal nerve: (1) hamstrings; (2) gastrocnemius; (3) tibialis posticus; (4) flexor longus digitorum; (5) flexor longus hallucis; (6) intrinsic; (7) flexor brevis hallucis.

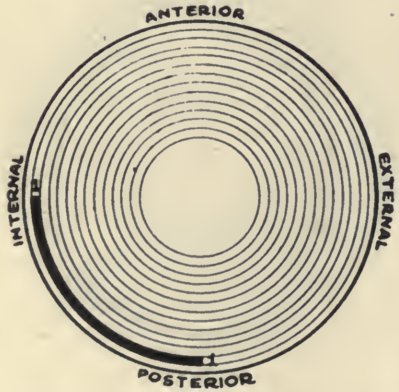

Figure 89

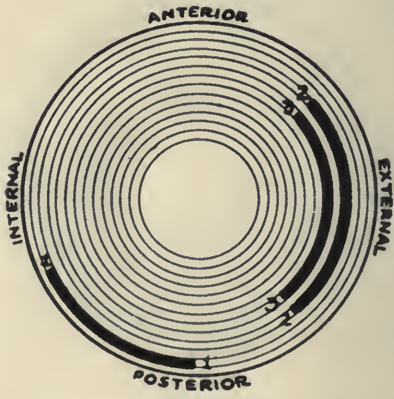

Figure 90

Fig. 89 (Case 10).—Abnormal external popliteal nerve: (1) peronei.

Fig. 90 (Case 14).—Abnormal external popliteal nerve: (1) peronei; (2) tibialis anticus; (3) extensors of the toes.

(d) Flexor Longus Digitorum: Two cases; Cases 12 (Fig. 70), *20* (Fig. 66).

In these cases the fibers lie postero-internally.

(e) Flexor Longus Hallucis: Two cases; Cases 12 (Fig. 70), *20* (Fig. 66).

Fibers to this muscle were found in the same position as those to the flexor longus digitorum. Reaction of the intrinsic muscles of the foot was not obtained in any of the cases in which the trunk was stimulated.

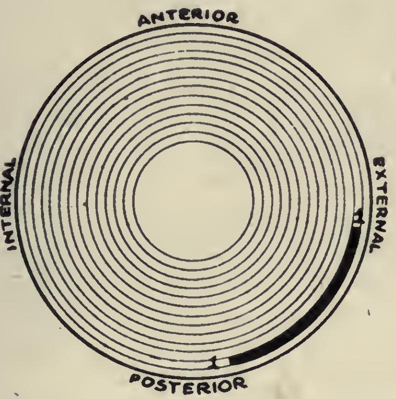

Figure 91

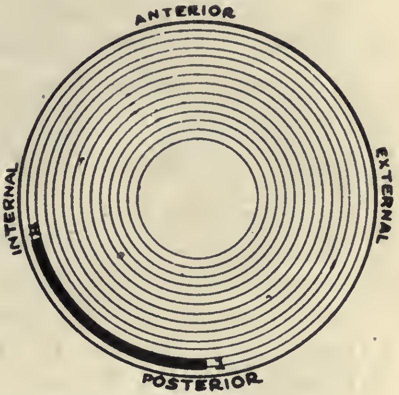

Figure 92

Fig. 91 (Case 46).—Abnormal external popliteal nerve: (1) peronei.
Fig. 92 (Case 38).—Abnormal external popliteal nerve: (1) peronei.

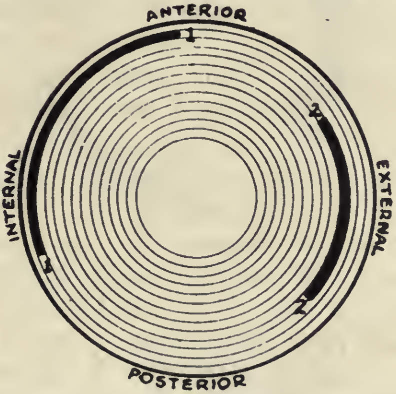

Figure 93

Fig. 93 (Case 59).—Abnormal external popliteal nerve: (1) peronei; (2) tibialis anticus.

2. *External Popliteal Division.*—Three cases; Cases 14 (Fig. 71), 21 (Fig. 72), 59 (Fig. 73).

(*a*) Peronei: Three cases; Cases 14 (Fig. 71), 21 (Fig. 72), 59 (Fig. 73).

These muscles lie posteriorly, external to the midline dividing the trunk into its divisions.

(*b*) TIBIALIS ANTICUS: Two cases; Cases 14 (Fig. 71), 59 (Fig. 73).

This muscle was stimulated twice from the trunk of the sciatic; its fibers lay in the same position as those of the peronei, i. e., postero-externa, in Case 14, and anterio-external in Case 59.

(*c*) EXTENSORS OF THE TOES AND GREAT TOE: One case; Case 14 (Fig. 71).

The position was the same as for the peronei. Figure 74 shows the composite results of stimulations of the sciatic nerve.

7. *Internal Popliteal Nerve.*—Twelve cases; Cases 1 (Fig. 75), 6 (Fig. 79), *7* (Fig. 85), 10 (Fig. 80), *40* (Fig. 76), 51 (Fig. 86), 52 (Fig. 82), 54 (Fig. 87), *56* (Fig. 77), 58 (Fig. 83), 63 (Fig. 78), 68 (Fig. 84).

(*a*) HAMSTRING MUSCLES: One case; Case 63 (Fig. 78).

The position is internal. This position corresponds to that found in four cases stimulated in the trunk. Stoffel also gives it as internal.

(b) GASTROCNEMIUS AND SOLEUS: Nine cases; Cases 1 (Fig. 75), *7* (Fig. 85), 10 (Fig. 80), *40* (Fig. 76), 52 (Fig. 82), *56* (Fig. 77), 58 (Fig. 83), 63 (Fig. 78), 68 (Fig. 84).

In Case 1, the fibers reacted anteriorly. In Cases *7*, 10, *40*, *56*, 58, 63 and 68, they reacted posteriorly; in case 52, on the internal side from the anterior to the posterior poles. With one exception, therefore, and this was the first case tried, the fibers were located in the same position, posteriorly. Marie, Meige and Gosset, and Stoffel found them posteriorly, Déjerine and Mouzon placed them internally.

(*c*) TIBIALIS POSTICUS: Ten cases; Cases 1 (Fig. 75), 6 (Fig 79), *7* (Fig. 85), 10 (Fig. 80), *40* (Fig. 78), 54 (Fig. 87), *56* (Fig. 77), 58 (Fig. 83), 63 (Fig. 78), 68 (Fig. 84).

The position in which this muscle could be stimulated varied considerably. We have no adequate explanation for this.

Cases 1 (Fig. 75), *7* (Fig. 85) internal reaction. Cases 10 (Fig. 80), *40* (Fig. 76), 54 (Fig. 87) anterior reaction. Case 6 (Fig. 79) posterior reaction. Cases *56* (Fig. 77), 58 (Fig. 83), *64* (Fig. 69) postero-internal reaction. Case 63 (Fig. 78) postero-external reaction. Case 68 (Fig. 84) antero-external reaction.

Marie, Meige and Gosset found that these fibers were antero-internal; Stoffel, antero-external; and Déjerine and Mouzon, external.

(d) FLEXOR LONGUS DIGITORUM: Five cases; Cases 6 (Fig. 80), *7* (Fig. 85), 10 (Fig. 80), *40* (Fig. 76), 54 (Fig. 87).

Cases 6, 10 and *40* reacted internally, Case 7, antero-internally and Case 54 posteriorly. Therefore, in four cases the position was internal or antero-internal and in one, posterior. The last position appears to be incorrect in view of the isolation and the position of the next muscle. The fasciculi of these two muscles would reasonably lie together. Marie, Meige and Gosset give this as postero-internal, Déjerine and Mouzon as external and Stoffel as antero-internal.

(e) Flexor Longus Hallucis: Four cases; Cases 6 (Fig. 80), *7* (Fig. 85), 10 (Fig. 80), *40* (Fig. 76).

This muscle was found internally or postero-internally in each case. Marie, Meige and Gosset found its position to be postero-internal, and Déjerine, Mouzon and Stoffel found it external.

(*f*) Intrinsic Muscles of the Foot: Two cases; Cases 51 (Fig. 87), 68 (Fig. 84).

Fibers to the plantar muscles, interossei and muscles of the small toe lay internally, while those to the flexor brevis digitorum lay postero-externally. Figure 88 shows results of stimulation of internal popliteal nerve.

8. *External Popliteal Nerve.*—Five cases; Cases 10 (Fig. 89), 14 (Fig. 90), 38 (Fig. 92), 46 (Fig. 91), 56 (Fig. 93).

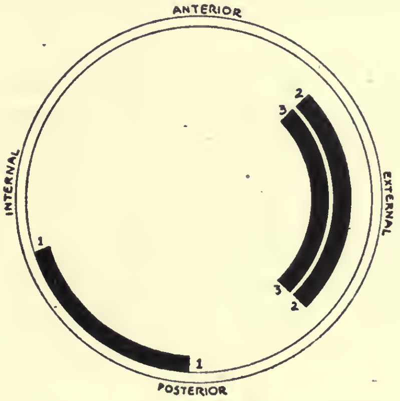

Figure 94

Fig. 94.—Results of stimulation of the external popliteal nerve: (1) peronei; (2) tibialis anticus; (3) extensors of the toes.

(*a*) Peronei: Five cases; Cases 10 (Fig. 89), 14 (Fig. 90), 38 (Fig. 92), 46 (Fig. 91), 59 (Fig. 93).

The fibers were found postero-internally in Cases 10, 14 and 38. In Case 46, in which the nerve was greatly damaged and but four or five bundles remained, the position was postero-external. The results obtained in the trunks agree with these. The apparent difference between the position is explained by the splitting of the sciatic trunk. Thus this group of fibers assumes a postero-internal position in the external popliteal nerve and a postero-external position in the trunk. Marie, Meige and Gosset placed them in a posterior position, Déjerine and Mouzon in an external position, and Stoffel in a postero-internal position.

(*b*) Tibialis Anticus: Two cases; Cases 14 (Fig. 90), 59 (Fig. 93).

These fibers lay externally. Marie, Meige and Gosset found their position antero-internal, Déjerine and Mouzon, internal and Stoffel, external.

(*c*) Extensors of the Toes: One case; Case 14 (Fig. 90).

These fibers lay externally. Marie, Meige and Gosset placed them antero-externally, Déjerine and Mouzon, internally and Stoffel externally. Figure 94 shows composite results of stimulation of the external popliteal nerve.

REPORT OF CASES

For purposes of brevity the following abbreviations are used in the case reports presented:

Lieutenant-Colonel Frazier, F.
Major Coleman, C.
Abductor pollicis, Ab. Pol.
Adductor pollicis, Ad. Pol.
Biceps, Bi.
Deltoid, Del.
Faradic, F.
Flexor brevis hallucis, F. B. H.
Flexor carpi radialis, F. C. R.
Flexor carpi ulnaris, F. C. U.
Flexor longus digitorum, F. L. D.
Flexor longus hallucis, F. L. H.
Flexor longus pollicis, F. L. P.
Flexor profundus digitorum, F. P. D.
Flexor sublimis digitorum, F. S. D.
Galvanic, G.
Gastrocnemius, Gast.
Hamstrings, Ham.
Major King, Kg.
Captain Knapp, Kp.
Hypothenar, Hr.
Interossei, Io.
Lumbricales, Lum.
Operation, Op.
Palmaris longus, P. L.
Pectoralis major, P. M.
Peronei, Per.
Pronator quadratus, P. Q.
Pronator radii teres, P. R. T.
Soleus, Sol.
Supinator longus, S. L.
Tibialis anticus, T. A.
Tibialis posticus, T. P.
Triceps, Tri.
Voluntary movement, V. M.
Wounded, Wd.

Case 41.[9]—G. B. (Figs. 4, 5, 6); wd. July 16, 1918; op. June 23, 1919, F. Diag.: Complete interruption of the fifth and sixth roots of the brachial plexus. Nerves Stimulated: Seventh and eighth cervical and first dorsal roots. Muscles Reacting: P. M., Tri. and flexors of the wrist, fingers and thumb. V. M.: Weak external rotation of the arm: no flexion of the forearm; otherwise normal.

Case 5.—A. C. (Figs. 7, 8); wd. Sept. 29, 1918; op. May 13, 1919, F. Diag.: Partial paralysis of the left brachial plexus, the cords of which were embedded in scar tissue 4 cm. below the clavicle. Nerves Stimulated: Posterior and lower cords of the brachial plexus. Muscles Reacting: Tri., Del., S. L., through the posterior cord, and the flexors of the index and middle fingers through the lower cord. Elec. Reaction (before op.): F.; none in the affected muscles. G.: slow contractions without polar reversal. V. M.: Flexion of the fingers and adduction of the thumb were the only movements observed.

Note.—This is an example of the phenomenon of difference between direct and percutaneous stimulation, in that the muscles which reacted at operation failed to do so at the previous clinical examination.

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[9] Sequence of cases as on Figures 2 and 3.

Case 31.—M. P. A. (Figs. 9, 10, 39, 59, 61); wd. July 18, 1918; op. June 5, 1919, F. Diag.: Lesion of the right musculospiral, ulnar and lower head of the median nerves, with neuromas of the musculospiral and ulnar. Nerves Stimulated and Muscles Reacting: circumflex, deltoid, musculospiral, Tri., S. L. and E. C. R.; median (outer head) P. R. T. (inner head), F. C. R., F. P. D., Ab. Pol.; ulnar, F. C. U., F. P. D. Elec. Reac.: F.: Reactions present in Tri., P. R. T., F. C. R., F. S. D., F. P. D. None in S. L., E. C. R. or F. C. U. G.: Slow reactions without reversal in all of the above muscles. V. M.: Weak in Opp. and Abd. P., absent in other affected muscles.

Note.—In this case the phenomenon of difference was obtained in several muscles: S. L., E. C. R. and F. C. U. The level of stimulation is not shown in the table, but was about the same as in Case 11.

Case 11.—H. J. M. (Figs. 11, 40); wd. Sept. 12, 1918; op. May. 21, 1919, F. Diag.: Lesion of the left median, ulnar and lesser internal cutaneous nerves; outer head of median involved in scar tissue. Nerves Stimulated and Muscles Reacting: ulnar: F. C. U., F. P. D.; median (inner head): F. C. R., P. L., F. L. D. and F. L. P.

Case 45.—H. V. (Figs. 13, 42); wd. Sept. 27, 1918; op July 23, 1919, C. Diag.: Partial paralysis of the median and ulnar nerves; neuromata. Nerves Stimulated and Muscles Reacting: ulnar: F. C. U., F. P. D.; median: P. R. T., F. P. D.

Case 2.—E. E. (Fig. 14); wd. July 18, 1918; op. May 5, 1919, F. Diag.; Complete lesions of the musculospiral and median nerves. Nerve Stimulated: median. Muscle Reacting: P. R. T. Elec. Reac. F.: none. G.: slow contraction with polar reversal in median muscles. V. M.: none.

Note.—Phenomenon of difference was obtained in this case in the P. R. T.

Case 43.—W. S. (Figs. 15, 43); wd. July 31, 1918; op. June 29, 1919, C. Diag.: Partial lesion of the median and ulnar nerves. Lateral neuroma of the median and causalgia. Nerves Stimulated and Muscles Reacting: median, P. R. T., F. C. R., F. P. D. and F. L. P.; ulnar, F. C. U., F. P. D., Ad. Pol. and Hr. V. M. present in all muscles reacting. E. R.: F. absent in all of the flexors of the fingers.

Note.—This case shows the phenomenon of difference in the long flexors of the fingers.

Case 16.—A. G. (Figs. 16, 41); wd. July 18, 1918; op. May 24, 1919, C. Diag.: Partial paralysis of the left median nerve. Nerves Stimulated and Muscles Reacting: median: F. C. R.; ulnar: Hr., I., Ad. Pol., F. C. U. and F. P. D. V. M. present in all muscles reacting.

Case 36.—L. I. (Fig. 17); wd. Sept. 28, 1918; op. June 10, 1919. Diag.: Complete paralysis of the left median and ulnar nerves, lateral neuromas on both. The P. R. T. reacted through the median at op.

Note.—This case shows the phenomenon of difference in the P. R. T.

Case 44.—D. P. (Fig. 18); wd. Oct. 8, 1918; op. July 19, 1919. Diag.; Neuroma of the median and compression of the ulnar nerves. Nerve Stimulated: median. Muscles Reacting: P. R. T., F. S D. and F. C. R.

Case 13.—G. C. K. (Figs. 19, 44); wd. Oct. 8, 1918; op. May 22, 1919, F. Diag.: Paralysis of the left ulnar and median nerves (incomplete in the latter), both nerves embedded in scar tissue. Nerves Stimulated and Muscles Reacting: median, P. R. T., F. C. R., P. L. and F. P. D.; ulnar, F. P. D. V. M.: limited to pronation and flexion of the wrist. E. R.: F.; there were no reactions previous to operation, in any of the ulnar muscles, and none in the median below the palmaris longus. G.: slow reaction without polar reversal in F. P. D. and F. C. U. and with reversal in the intrinsic hand muscles.

Note.—Stimulation of the ulnar exposed at op. in this case gave a reaction in F. C. U. when the nerve was stimulated below the lesion, but not from above it—a direct paradoxical phenomenon.

Case 3.—C. W. (Figs. 20, 46); wd. Oct. 4, 1918; op. May 7, 1919, C. Diag.; Partial paralysis of the ulnar nerve. Nerves Stimulated and Muscles Reacting: median, P. R. T., F. C. R. and F. L. P.; ulnar, F. C. U. and F. P. D. V. M.: included slight movement in the F. C. U. E. R.: F.; no reaction in ulnar distribution. G.: slow contractions with polar reversals in the ulnar muscles.

Note.—This case shows the phenomenon of difference between direct and percutaneous stimulation in the F. C. U. and F. P. D. The case is also interesting as showing the predominance of reactions in the proximal muscles of a normal nerve, the median.

Case 26.—T. F. (Fig. 21); wd. July 20, 1918; op. May 31, 1919, C. Diag.: Partial paralysis of the left median nerve, lateral neuroma. Nerve Stimulated and Muscles Reacting: median, P. R. T., F. C. R., F. L. P. and F. P. D.

Note. — The phenomenon of difference between direct and percutaneous stimulation was demonstrated in this case, which is also of special interest since it was possible to obtain reactions of the P. R. T. from the external side of the nerve above the lesion, while an inch or so below a similar reaction was obtained from the inner aspect of the nerve, indicating that the lesion lay just at the point of crossing of the fibers supplying the pronator.

Case 9.—M. P. H. (Fig. 22); wd. July 15, 1918; op. May 5, 1919, F. Diag.: Paralysis of the median and ulnar nerves; both were embedded in scar tissue. Nerve Stimulated: median, muscles reacting, F. C. R., F. P. D., P. R. T. and F. L. P. V. M.: limited to contractions of the P. R. T. and F. C. R. E. R.: F.; no reactions. G.: slow reactions with polar reversal in the muscles affected.

Note.—The phenomenon of difference was demonstrated in this case.

Case 67.—C. K. (Fig. 23); wd. Oct. 19, 1918; op. Aug. 21, 1919, Kg. Diag.: Paralysis of the left median nerve. The median nerve was stimulated and the muscles reacting were the P. R. T. and F. C. R.

Case 50.—W. E. T. (Figs. 24, 47); wd. Oct. 16, 1918; op. July 30, 1919, Kg. Diag.: Paralysis of the left median and ulnar nerves, both of which were embedded in scar tissue. Nerves Stimulated and Muscles Reacting: median, P. R. T., F. C. R. and F. P. D.; ulnar, F. C. U.

Case 53.—F. H. M. (Fig. 25); wd. Sept. 27, 1918; op. Aug. 4, 1919, Kp. Diag.: Paralysis of the left median nerve. This nerve was stimulated with resulting reactions in the P. R. T., F. C. R. and F. P. D. muscles.

Case 19.—F. E. C. (Figs. 26, 27, 28); wd. Nov. 1, 1918; op. May 27, 1919, F. (local anesthesia). Diag.: Paralysis of the right median and ulnar nerves; causalgia of the median. The median nerve was stimulated at operation, giving reactions in P. Q., O. P. Ab. Pol., first and second Lum. The sensory reaction was described by the patient by the words "it hurts" and was localized in all the fingers except the fifth.

Case 30.—W. B. (Fig. 29); wd. July 30, 1918; op. June 4, 1919. Diag.: Paralysis of the right median nerve below the branches to the forearm muscles. Nerve Stimulated and Muscles Reacting: median, F. C. R. and P. L. The stimulation was applied below the point of offset of the branch to the P. R. T., which was seen leaving the nerve from its inner side.

Case 69.—S. K. (Fig. 30); wd. Sept. 2, 1918; op. Sept. 30, 1919, Kg. Diag.: Partial paralysis of the right median nerve. This nerve was stimulated at operation and reactions obtained in the P. Q. and F. P. L. muscles.

Case 27.—J. T. W. (Figs. 31, 32, 33, 57); wd. Nov. 19, 1918; op. June 19, 1919; local anesthesia. Diag.: Partial paralysis of the median and ulnar nerves; neuromas. Only sensory reactions were obtained.

Case 17.—D. D. (Figs. 34, 35, 36); wd. Oct. 1, 1918; op. May 26, 1919, C., under local anesthesia. Diag.: Paralysis of the median nerve at the wrist; anatomic division with proximal neuroma. Only sensory reactions were obtained, the patient complaining of "tingling" and "burning" when the proximal stump was stimulated.

Case 60.—J. J. (Fig. 45); wd. Oct. 10, 1918; op. Aug. 8, 1919, Kg. Diag.: Partial paralysis of the right ulnar nerve; neuroma. Reaction was obtained in the F. C. U. muscle.

Case 66.—P. F. (Fig. 48); wd. Oct. 18, 1918; op. Aug. 19, 1919, Kp. Diag.: Partial paralysis of the left ulnar nerve. Reactions were obtained in the F. C. U., F. P. D., Io., Ad. Pol. and Ab. M. D.

Case 42.—W. G. (Fig. 49); wd. Oct. 18, 1918; op. June 25, 1919, C. Diag.: Partial paralysis of the left ulnar nerve; neuroma Muscles reacting: F. P. D. and F. C. U.

Case 28.—E. P. (Fig. 50); wd. Nov. 3, 1918; op. June 3, 1919, F. Diag.: Compression of the left ulnar nerve below branches to the forearm muscles. Reactions were obtained in the Ad. Pol. and Io. from stimulation below but not from above the lesion on the exposed nerve.

Note.—An example of the direct paradoxical phenomenon.

Case 8.—P. B. (Fig. 51); wd. Sept. 26, 1918; op. May 15, 1919, C. Diag.: Complete lesion of the right ulnar nerve below the branch to the F. C. U.; separation of 3 cm. with proximal and distal neuromas. Muscles reacting F. C. U. and F. P. D. E. R.: F.; reactions in the ulnar distribution limited to the F. C. U. G.; F. P. D. gave a slow reaction without polar reversal.

Note.—The reactions were obtained by stimulating the branches to the muscles, not the main trunk. These branches left the nerve from its internal aspect. The case shows the difference between direct and percutaneous stimulation in the reactions of the F. P. D.

Case 47.—J. F. B. (Fig. 52); wd. Oct. 14, 1918; op. July 15, 1919, C. Diag.: Partial paralysis of the left ulnar nerve; only a small fasciculus on the inner border of the nerve was not severed. Reactions obtained in the Io.

Case 49.—A. F. J. (Fig. 53); wd. June 19, 1918; op. June 29, 1919, F. Diag.: Partial paralysis of the left ulnar nerve; fusiform neuroma at the site of a neurorrhaphy performed Dec. 3, 1918. (The second operation was done because there was no improvement after the first.) V. M.: none in any of the ulnar hand muscles. E. R.: F.; no reaction in ulnar hand muscles. The intrinsic muscles of the hand supplied by the ulnar nerve reacted from stimulation of the exposed nerve at operation.

Note.—This example of the phenomenon of difference occurred eight months after complete division and suture of a nerve, and must have been due to partial regeneration which had not shown itself in a return of voluntary movement.

Case 18.—M. C. M. (Fig. 54); wd. July 20, 1918; op. May 23, 1919, F. Diag.: Lesion of the musculospiral nerve. No reactions were obtained from this nerve, but the ulnar nerve which was normal was exposed and stimulated. The muscles reacting were: Ad. Pol., F. C. U. and F. P. D.

Cases 34 and 35.—(Figs. 55, 56, 57). These cases are included since in the former the branches to the F. C. U. and F. P. D., and in the latter branches to the F. C. U. were clearly seen leaving the ulnar nerve from the inner side. They were stimulated in each instance, confirming the observations.

Case 39.—J. W. W. (Fig. 60); wd. Nov. 1, 1918; op. June 19, 1919, F. Diag.: Paralysis of the inner cord of the brachial plexus. The circumflex nerve was stimulated at operation, the deltoid muscle reacting. V. M.: slight in the deltoid. E. R.: F.; none in deltoid. G.; slow wavy without polar reversal in the deltoid.

Note.—This case showed the phenomenon of difference between direct and percutaneous stimulation.

Case 33.—G. R. C. (Fig. 62); wd. Nov. 5, 1918; op. June 6, 1919, C. Diag. paralysis of the musculospiral nerve below branches to the triceps; neuroma. Nerve Stimulated: musculospiral. Muscle Reacting: triceps.

Case 15.—E. D. (Fig. 63); wd. Aug. 6, 1918; op. May 23, 1919, F. Diag.: Paralysis of the musculospiral nerve below branch to triceps; neuroma. Nerve Stimulated: musculospiral. Muscle Reacting: extensor indicis. V. M.: none in musculospiral distribution. E. R.: F.; none. G.; slow reaction without polar reversal.

Note.—Similar responses were obtained at operation by stimulating the nerve above and below the lesion, both before and after section of the nerve. The case is an example of the phenomenon of difference between direct and percutaneous stimulation.

Case 22.—A. J. B. (Fig. 64); wd. Oct. 6, 1918; op. May 29, 1919, F. Diag.: Compression of the right musculospiral nerve below the branch to the E. C. R. Nerve Stimulated: musculospiral. Muscle Reacting: E. C. R.

Case 20.—R. M. S. (Fig. 66); wd. Oct. 14, 1918; op. May 27, 1919, C. Diag.: Paralysis of the left external popliteal nerve; neuroma. Nerve Stimulated: Ext. Pop. Muscles Reacting: H. S., Gast., F. L. H., F. L. D. and T. P.

Case 23.—F. S. (Fig. 67); wd. Sept. 29, 1918; op. April 4, 1919, C. Diag.: Paralysis of the left sciatic nerve; neuroma. Nerve Stimulated: sciatic. Muscle Reacting: H. S.

Case 24.—W. H. H. (Fig. 68); wd. July 18, 1918; op. April 22, 1919, F. Diag.: Paralysis of the right sciatic nerve, partial; lateral neuroma on outer side. Nerve Stimulated: sciatic. Muscles Reacting: Gast. and Sol. group. V. M., slight flexion of great and fifth toes; E. R. F., none below knee; G., wavy contraction without reversal in internal popliteals, and with reversal in external popliteal muscles.

Note.—We are indebted to Lieutenant-Colonel Frazier for the notes on this case. It is an example of the phenomenon of difference between direct and percutaneous stimulation as the gastrocneumeus and soleus reacted from direct stimulation of the nerve at operation, but had failed to react from stimulation through skin.

Case 64.—B. (Fig. 69); wd. Sept. 27, 1918; op. Aug. 16, 1919, Kg. Diag.: Partial paralysis of the external division of the left sciatic nerve; neuroma. Nerve Stimulated: sciatic (Int. Pop. division). Muscles Reacting: Bi., Gast., Sol and T. P.

CASE 12.—A. G. (Fig. 70); wd. Aug. 9, 1918; op. May 21, 1919, C. Diag.: Partial paralysis of Int. and Ext. Pop. nerves. Nerve Stimulated: Int. Pop. Muscle Reacting: Gast. and Sol. group, T. P., F. L. D. and F. L. H.

CASE 14.—S. H. (Figs. 71 and 90); wd. Sept. 29, 1918; op. May. 22, 1919, C. Diag.: Paralysis of the right sciatic nerve; anatomic division of the Int. Pop. portion of the nerve. Nerve Stimulated: Ext. Pop. portion of the sciatic. Muscles Reacting: T. A., Per. and extensors of the toes. V. M.: slight movement of the muscles supplied by the Ext. Pop. nerve. E. R. F.; weak reactions in same muscles. G.: slow reaction without polar reversal.

Note.—This case is interesting in showing the difference in position of the fasciculi which supply the peronei in the trunk of the sciatic and in that of the Ext. Pop. nerve. They lie on the posterior-internal aspect of the nerve, which only permits stimulation from the posterior aspect of the sciatic, but when the external division is separated these fasciculi can be stimulated both posteriorly and internally.

CASE 21.—L. E. (Fig. 72); wd. Sept. 29, 1918; op. May 28, 1919; C. Diag. Partial paralysis of the left sciatic nerve; neuroma on int. div. Nerve Stimulated: Ext. Pop. div. of sciatic. Muscles Reacting: peronei. V. M.: none. E. R.: F.: none. G.: slow reaction without polar reversal.

Note.—This is another example of the phenomenon of difference between direct and percutaneous stimulation.

CASE 59.—J. J. (Figs. 73, 93); wd. July 20, 1918; op. Aug. 8, 1919, Kg. Diag.: Incomplete paralysis of the left sciatic nerve; causalgia; compression by scar tissue. Nerve Stimulated: sciatic (ext. portion). Muscles Reacting: Per. and T. A.

Note.—This case is of interest in showing the apparent rotation of the peronei fasciculi. The results of stimulation of the external popliteal alone differ somewhat from those in Case 14. However, the former stimulation was applied at a point above the bifurcation after the two divisions of the sciatic had been dissected apart, while in this case the stimulation was applied near the head of the fibula. The nerve makes a twist at about this place which probably accounts for the seeming inconsistency.

CASE 1.—F. M. (Fig. 75); wd. Sept. 30, 1918; op. April 29, 1919, F. Diag.: Partial paralysis of the right sciatic nerve; neuroma of the external portion. Nerve Stimulated: Int. Pop. division of the sciatic. Muscles Reacting: Gast. and Sol. group and T. P.

CASE 40.—A. L. R. (Fig. 76); wd. Aug. 4, 1918; op. June 19, 1919, A. Diag.: Paralysis of the left external popliteal nerve; neuroma. Nerve Stimulated: internal popliteal. Muscles Reacting: Gast. and Sol., T. P., F. L. D. and F. L. H.

CASE 56.—G. G. (Fig. 77); wd. Sept. 29, 1918; op. Aug. 6, 1919, C. Diag.: Incomplete paralysis of the left sciatic nerve; external division embedded in scar. Nerve Stimulated: internal popliteal. Muscles Reacting: Gast. and Sol., T. P.

Note.—The internal division was separated before stimuli were applied.

CASE 63.—M. F. (Fig. 78); wd. Oct. 2, 1918; op. Aug. 14, 1919, Kg. Diag.: Incomplete paralysis of the right sciatic nerve; anatomic interruption of the external portion. Nerve Stimulated: internal popliteal. Muscles Reacting: Gast. and Sol., Bi. and T. P.

Note.—The internal division was separated before stimuli were applied.

Case 6.—F. S. (Fig. 79); wd. Sept. 27, 1918; op. May. 4, 1919, C. Diag.: Partial paralysis of the right sciatic nerve; complete division of the external popliteal and neuroma of the internal popliteal, external aspect. Nerve Stimulated: internal popliteal. Muscles Reacting: T. P., F. L. D. and F. L. H.

Case 10.—D. H. (Figs. 80, 81, 89); wd. Sept. 30, 1918; op. May 17, 1919, F. Diag.: Partial paralysis of the right sciatic nerve; neuroma of the external division. Nerve Stimulated: internal popliteal, external popliteal. Muscles Reacting: Per., Gast. and Sol., T. P., F. L. D. and F. L. H.

Note.—This case is interesting in that the external popliteal nerve was split and stimulation was applied to the cut surface. The results showed that the fasciculi to the peronei lay posteriorly.

Case 52.—N. G. (Fig. 82); wd. July 24, 1918; op. Aug. 4, 1919, C. Diag.: Partial paralysis of the right sciatic nerve; neuroma of the external popliteal and external side of the internal popliteal nerves. Nerve Stimulated: internal popliteal. Muscles Reacting: Gast. and Sol.

Case 58.—R. M. L. (Fig. 83); wd. Oct. 23, 1918; op. Aug. 7, 1919, Kg. Diag.: Paralysis of the left sciatic nerve; interruption of external division. Nerve Stimulated: internal popliteal. Muscles Reacting: Gast. and Sol., and T. P.

Case 68.—J. G. (Fig. 84); Diag.: Partial paralysis of the sciatic nerve. V. M.: slight in muscles of the distribution of the internal popliteal nerve. Nerve Stimulated: internal division of the sciatic. Muscles Reacting: Gast. and Sol., T. P. and intrinsic muscles of the foot.

Case 7.—O. C. S. (Fig. 85); wd. Aug. 4, 1918; op. May 14, 1919, F. Diag.: Paralysis of the left external popliteal nerve; anatomic division. Nerve Stimulated: internal popliteal. Muscles Reacting: Gast. and Sol., T. P., F. L. D and F. L. H.

Case 51.—S. S. (Fig. 86); wd. Oct. 10, 1918; op. July 30, 1919. Diag.: Paralysis of the internal popliteal nerve; compression by scar tissue. Nerve Stimulated: internal popliteal. Muscles Reacting: intrinsic muscles of the foot.

Case 54.—A. S. T. (Fig. 87); wd. Sept. 28, 1918; op. Aug. 5, 1919, Kp. Diag.: Partial paralysis of the internal popliteal nerve; neuroma; causalgia. Nerve Stimulated: internal popliteal Muscles Reacting: T. P. and F. L. D.

Case 46.—F. O. (Fig. 91); wd. Aug. 3, 1918; op. July 24, 1919. Diag.: Partial paralysis of the right external popliteal nerve; neuroma involving the nerve with the exception of a few fasciculi on the posterior-external aspect. Nerve Stimulated: external popliteal. Muscles Reacting: the peronei.

Note.—The results in this case do not correspond to the others for this group of muscles, probably because the nerve was so extensively involved and may have been rotated by the injury.

Case 38.—J. R. (Fig. 92); wd. Oct. 9, 1918; op. June 17, 1919. Diag.: Paralysis of the left anterior tibial nerve; lateral neuroma. Nerve Stimulated: anterior tibial. Muscles Reacting: peronei.[10]

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[10] In addition to the references already given, the following may be of interest:

Sherren: Injuries of Nerves and Their Treatment, New York, William Wood & Co., p. 196.

Platt and Brentnall: Faradic Stimulation of Nerve and Muscle During Operations, Lancet **1**:884 (May 24) 1919.

SOME OBSERVATIONS ON EPILEPTICS AND ON EPILEPSY, CHIEFLY FROM A ROENTGEN-RAY STANDPOINT

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At the fortieth annual meeting of the American Neurological Association, held in May, 1914, a paper was presented, entitled "Observations on Epilepsy Chiefly from an X-Ray Standpoint," by Drs. T. M. T. McKennan, George C. Johnston and C. H. Henninger. In that paper it was shown that there were apparently deposits of bone in the base of the skull in a large percentage of epileptics. An endeavor was made to show the way in which this deposit occurred and that, together with this deposit, there was probably a crippling of the pituitary gland. To make this clear I quote:

> The interpretation of the changes found at the base of the skull in the essential epilepsies at first seemed to us to be very difficult. Our first idea was that it had an inflammatory origin. This we were forced to abandon for the reason that we could find no cause of an infective character that would produce inflammation, for the relative regularity of the changes in certain situations in the cases examined. The viewpoint that the bony overgrowth is a local acromegaly and that the bony deposit is brought about through venous congestion at the base of the skull seems to us to be the only logical explanation.
>
> To comprehend this a knowledge of the venous circulation at the base of the skull is essential. The veins from the anterior lobe of the pituitary gland empty into the intercavernous sinuses and the secretion from the anterior lobe of the pituitary is poured into these sinuses. The intercavernous sinuses vary in number from one to three; there is probably an average of two. Knott found a single intercavernous sinus in twelve out of forty-four examined. In fifteen out of forty-four he found two. Most textbooks of anatomy give these two intercavernous sinuses the name of the circular sinus. It should, however, be noted that the intercavernous sinuses are really veins and not sinuses. Hence they are collapsible.
>
> These intercavernous veins serve to join the two cavernous sinuses and they receive also a number of small veins from the adjacent parietal dura mater. This dura, as we know, is the internal periostium. These dural veins drain an area of the base of the skull comprising the clinoid processes and the dorsum sella, the anterior fossa and the body of the sphenoid. The anastomosis of these veins with other veins probably varies considerably. The bony deposit in epileptics corresponds to the areas drained by these dural veins of the base and leads directly to the conviction that the process of bony change can only be brought about by some pressure within the small confined boxlike space that contains the pituitary gland as well as the intercavernous veins.
>
> Pressure within this boxlike space that would prevent the intercavernous veins from discharging their contents into the cavernous sinuses would cause

the blood in this boxlike structure to be backed into the dural veins. The circulation at this point would be reversed and the blood loaded with secretion from the anterior lobe of the pituitary gland would bathe the bony structure of the base of the skull in the region of the clinoid processes, dorsum sella, the anterior fossa and the body of the sphenoid bone. Increased bony deposit would consequently be the result. This deposit, however, would vary in amount depending on two factors; first, the duration of pressure that causes the reversal of circulation, and second, on the freedom of anastomosis of the dural veins.

The next question that logically arises is: What can cause the pressure within this boxlike structure? Hypertrophy of the pituitary gland is improbable for this condition has not been found in postmortem examinations of epileptics. It seems more probable to us that it is intermittent hyperemia, and this viewpoint is somewhat confirmed by the fact that postmortem examinations of epileptics reveal abnormally large veins within the pituitary gland.

The question then naturally arises: What is the cause, or what can be the cause of intermittent hyperemia in this situation? Does it occur from the attack of epilepsy with the attendant general venous congestion? We think this extremely improbable, for our cases of symptomatic epilepsy showed no bony deposit. Again, hyperemia, the result of an attack of epilepsy, would naturally last for a few minutes only and the blood stream would soon return to its normal course. Hyperemia would have to exist for hours or days to permit of any bony deposit.

It seems more probable that the venous hyperemia would be due to some vasomotor disturbance of the anterior lobe of the pituitary gland. This same venous hyperemia would doubtless tend to prevent a proper amount of secretion of the posterior lobe from gaining access to the third ventricle by way of the infundibulum and in all probability repeated hyperemias would cause a degree of crippling of the entire pituitary gland, the posterior lobe, as well as the anterior lobe.

CASES STUDIED BY AUTHOR

The present study comprises ninety cases.

TABLE 1.—Results of the Roentgen-Ray Findings

	No. of Cases	Per Cent.
Tumors or evidence of pressure in the interpituitary area	9	10.
Bony deposits	52	57.77
Small areas	10	11.
Calcareous degeneration	2	2.5
Cerebropathy (this is not shown by the roentgen ray)	9	10.
No changes	8	8.8
Total	90	100.

The designation "small area" may be explained thus: In some cases the pituitary area appears to be very small. It has the appearance of being infantile, and we designate it a small or infantile area. It would be a strong argument in favor of a small pituitary gland within this area—a gland that possibly has not increased in size commensurately with bodily growth and, therefore, probably an inefficient gland. We cannot accurately measure this area and say definitely what size the area should be to be normal, but by experience with roentgen-ray plates we can judge what appears to be normal and what appears to be abnormal. In many cases with increased bony deposit I have noted a pituitary area that appears to be small, and it is my belief that an increasing number will be found to have abnormally small pituitary areas. As to the extent of the bony deposits or bony overgrowth, I am now quite satisfied that it may occur in almost any part of the skull and that the situation of the main deposit is governed entirely by the extent and kind of the venous anastomosis that exists in the individual case. I am coming to the belief that essential epilepsy is largely due to a crippling of the pituitary gland in one way or another. I believe that the most frequent cause is probably recurring hyperemia of the gland; that second in importance is a gland that has not evoluted properly—a small inefficient gland; and third in importance is pituitary tumor or struma, or in some cases probably simple hypertrophy. The question of abnormality of the pituitary gland in its relation to epilepsy is greatly illuminated by a consideration of cases of epilepsy originating in persons over 35 years of age. (These cases were taken from my total number of cases in which I have roentgen-ray plates).

TABLE 2.—CASES STARTING IN PERSONS OVER 35 YEARS OF AGE

	No. of Cases	Per Cent.
Tumor	13	48.1
Bony deposits	7	25.9
Small area	4	14.8
Cerebropathies	3	11.2
Total	27	100.

It is well known that tumor of the pituitary gland usually disturbs the function of that gland. Epilepsies in these cases are regarded as due to disturbance or destruction of the function of this gland. If we accept the theory that the small area is an evidence of pituitary inefficiency, and further, if we accept the theory that increased bony deposits are an evidence of pituitary crippling, it would be shown by the figures above that in cases of epilepsy originating in persons over 35 years of age, 88 per cent. present roentgen-ray evidence of pituitary inefficiency.

To view the matter from another angle, let us leave out of consideration the cases of cerebropathy for the time. Thus there are twenty-four cases of epilepsy starting in persons over 35 years of age. More than 50 per cent. of these cases being known to be due to an organic affection of the gland, causing it to be inefficient, would be a strong argument in itself for the conclusion that the other cases must have inefficient pituitary glands from some cause or causes.

TABLE 3.—Epilepsy Beginning in Persons Over 35 Years of Age

	No. of Cases	Per Cent.
Tumor	13	53.63
Bony deposits	7	29.87
Small area	4	16.50
Total	24	100.

In some cases in which there was evidence of pressure in the pituitary area I have been able to have a roentgenogram made again in one or more years and have, in some instances, found that there was no progression. It would seem that if these cases are struma or other tumor growths the process of enlargement had ceased. It is possible that some of them may be simple hypertrophy.

The question of pituitary feeding in epileptics, in my opinion, is an important one. I have found that the extract of the whole gland in 2 grain doses, three times a day, and given four hours after eating, is the most satisfactory. Bromids should always be used in conjunction with the pituitary extract. I am in the habit of using from 45 to 60 grains of bromid daily. After one year less bromid can be used, but the pituitary feeding must be kept up indefinitely, probably during the life of the patient.

CONCERNING THE COLLOIDAL MASTIC TEST *

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The reported results of the application of the colloidal mastic test to the examination of pathologic spinal fluids have been so contradictory that it has appeared wise to conduct an investigation to determine the degree of parallelism, in regard to their clinical value, between the colloidal gold and the colloidal mastic reactions, and the possible cause for a discrepancy, if any exists.

HISTORICAL NOTE

The application of colloidal solutions to the examination of the spinal fluid dates from the work of Lange,[1] who made use of a colloidal gold solution. Neisser and Friedemann,[2] among others, demonstrated the similarity in action between soluble proteins and a colloidal gold or mastic solution. Emmanuel,[3] in 1915, published his notes on the application of the colloidal mastic test to the examination of various pathologic spinal fluids. In brief, the technic used by Emmanuel in employing a colloidal mastic solution is as follows: One and five tenths c.c. of a 1.25 per cent. sodium chlorid solution is placed in the first of five tubes and 1 c.c. is placed in each of the remaining tubes. Five tenths c.c. of the spinal fluid to be tested is placed in the first tube, the contents are well mixed and 1 c.c. is transferred to the second tube and so on. The 1 c.c. of fluid removed from the fourth tube is discarded, the fifth tube serving as a control. One c.c. of the mastic emulsion (prepared as will be described) is now added to each tube, the tubes are shaken and allowed to stand for twelve hours. Abnormal fluids give a precipitation in varying degree in some or all of the tubes.

Of the thirty-two fluids examined by Emmanuel, precipitation occurred in all the dilutions prepared from the fluids of paretics, tabetics and cerebrospinal syphilitics. It was found that frequently

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[1] Lange, C.: Die Ausflockung Kolloidalen Goldes durch Cerebrospinalflüssigkeit bei leutischen Affektionem des zentralnervensystem, Ztschr. f. Chemotherap. **1**:44, 1912.

[2] Neisser, M., and Friedemann, O.: Studien über Ausflockungserscheinungen II, Beziehung zur Bakterienagglutination, München. med. Wchnschr. **51**:827, 1904.

[3] Emmanuel, G.: Eine neue Reaktion zur Untersuchungen des Liquor Cerebrospinalis, Berl. klin. Wchnschr. **52**:792, 1915.

there was some precipitation in the fourth tube, even with normal fluids. Jacobstahl and Kafka,[4] in 1918, described a slightly different technic, using two curves—depending on the salt sensitiveness of the mastic sol. For recording their results they made use of eight degrees of change in the mastic sol.

In regard to the diagnostic value of the test, one finds rather conflicting opinions. Victors,[5] using the original method of Emmanuel, reported that the technic of the test was relatively simple and that the information obtained from it was trustworthy. Lowrey [6] examined thirty-six fluids, using the original technic and decided "that it cannot obviously do the work of the gold test." Urechia and Jorgulesci [7] using the original technic decided that the mastic and the gold sol reactions gave the same results but that the mastic was not as sensitive as the gold.

In 1917, Cutting [8] reported that by the addition of a trace of potassium carbonate to the sodium chlorid solution, used for making the dilutions, the control tube showed no precipitation and the dilutions of the spinal fluid could be run out as far as desired. In the same year, Smith [9] reported the use of the Cutting modification of the test. He carried the dilution through ten tubes, but in particular, made use of a numerical nomenclature for expressing the degree of precipitation of the mastic: 1, denoted a milky fluid, loss of opalescence and a very slight precipitate; 2, a distinct precipitate but with a milky supernatant fluid; 3, a marked precipitate with a slight cloudiness of the fluid above; and 4, complete precipitation of all the colloid. Smith reported duplicate curves on the fluids of twenty-three different patients. His results show an exact agreement (as regards type of reaction) of the two curves in twenty-two of the twenty-three fluids examined. The gold curve of six of his fluids was paretic or near-paretic in type; in eleven fluids there was a change of the nonparetic variety; while six of the fluids yielded a negative result. Smith is of the opinion that the information obtained from the mastic test is the same as that obtained from the gold test. Immermann[10] in his report on the mastic test concluded that "the gum mastic test is not equivalent to nor supplementary to the colloidal gold reaction." His clinical material consisted of the fluids from twenty cases. If Smith's numerical nomenclature be applied to Immermann's description of his curves, and the gold and mastic curves be graphically plotted and compared, it will be found that with three of the fluids the curves were relatively identical, with five of the fluids they were similar in some respects, while with the remaining twelve fluids they were dissimilar.

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[4] Jacobstahl and Kafka: Mastix-Reaktion, Berl. klin. Wchnschr. **55**:249, 1918.

[5] Victors, E. A.: Colloidal Mastic Reaction of Cerebrospinal Fluid, California State J. M. **16**:32, 1918.

[6] Lowrey, L. G.: Mastiche and Potassium Permanganate Tests Applied to the Cerebrospinal Fluids of the Insane, Boston M. & S. J. **177**:557, 1917.

[7] Urechia, C. J., and Jorgulescu, N.: L'epreuve colloidale au mastic d'Emmanuel dans le liquide cephalo-rachidien, Compt. rend. Soc. de biol. **79**: 893, 1916.

[8] Cutting, J. A.: A New Mastic Test for the Spinal Fluid, J. A. M. A. **68**:1810 (June 16) 1917.

[9] Smith, E. R.: The Mastic Reaction on the Cerebrospinal Fluid, Med. Rec. **92**:675, 1917.

Smith and Lowrey,[11] after the experience resulting from the examination of 268 fluids by the Cutting modification of the Emmanuel test, felt that "the mastiche reagent is simple and the interpretation is easy. Furthermore, it seems to be very accurate." These workers, however, did not make use of a numerical system for recording their results but simply reported the mastic test as positive or negative. With no case of the 268 was the mastic test positive when the gold was negative.

Langdon,[12] after the examination of a series of fluids by both colloidal reactions, was of the opinion that the mastic test was "probably not as delicate or valuable" as the gold test. He stated that the gold test indicated whether the curve was of the paretic, syphilitic or meningitic type while the mastic test showed merely a positive or negative result. Langdon made use of no numerical method of recording the changes in the various tubes of the mastic test.

METHODS USED

Preparation of Mastic Emulsion.—Ten grams of gum mastic were dissolved in 100 c.c. of absolute alcohol. The resultant cloudy solution was filtered several times through paper, until a brilliant straw colored fluid was obtained. This solution was kept in glass stoppered bottles in the open laboratory, but never in the direct sunlight. The emulsion was prepared from this stock solution thus: To 1 c.c. of the stock solution in a test tube, 9 c.c. of absolute alcohol were added. Forty c.c. of distilled water were placed in a small Erlenmeyer flask and the mastic solution in alcohol was then rapidly poured in and the whole gently mixed by rotation.

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[10] Immerman, S. L.: The Mechanism and the Significance of the Gum Mastic Test, J. A. M. A. **69**:2027 (Dec. 15) 1917.

[11] Smith, C. E., and Lowrey, L. G.: On the Use of the Emmanuel-Cutting Mastiche Test in Examining Spinal Fluids from Psychopathic Subjects, Boston M. & S. J. **177**:557, 1917.

[12] Langdon, F.: The Mastic Test for the Diagnosis of Cerebrospinal Syphilis, J. Lab. & Clin. M. **3**:376, 1918.

Preparation of the Dilutions.—Ten small test tubes were set up in a rack. To the first were added 1.5 c.c. of the salt solution (99 c.c. of 1.25 per cent. sodium chlorid solution plus 1 c.c. of 0.5 per cent. potassium carbonate solution), and to each of the remainder 1 c.c. was added. Five tenths c.c. of the spinal fluid was added to the first tube, and after mixing, 1 c.c. was transferred to the second tube; this was mixed, and so on. To each tube 1 c.c. of the mastic emulsion was added; each tube was shaken just enough to secure even distribution; the tubes were allowed to stand over night and were then read. The results were expressed according to Smith nomenclature. The gold tests were carried out according to the usual standards.

COMPARISON OF THE GOLD AND MASTIC CURVES OF THE FLUIDS EXAMINED

The comparative curves presented in this paper are 100 in number and form a consecutive series of the fluids examined. These 100 fluids exhibited a paretic gold curve in sixteen instances, a positive gold curve but not of the paretic variety in fifty-five instances, and in twenty-nine instances gave a negative reaction. For comparative study the fluid findings have been tabulated in these three groups on the basis of the gold test.

TABLE 1.—The Mastic Reaction on Fluids Having a Paretic Type of Colloidal Gold Reaction

Fluid No.	Diagnosis	Gold	Mastic	Cell Count	Pandy Test	Cerebrospinal Fluid Wassermann Reaction
1	Paresis	5554344300	4444431000	34	+	++++
2	Paresis	5555320000	3444200000	60	..	++++
3	Paresis	5542000000	4431000000	36	..	++++
4	Syphilitic myelitis	5543100000	4431000000	14	+	++++
5	Paresis	4543100000	4432100000	12	+	++++
6	Paresis	5555420000	4444310000	33	+	++++
7	Paresis	5542100000	4432000000	6	+	++++
8	Hemiplegia	5554100000	2322000000	74	+	++++
9	Brain tumor	5555433210	4444443210	6	+	—
10	Lethargic encephalitis	5554320000	4444431000	6	+	—
11	Paresis	5555554310	4444310000	..	..	++++
12	Paresis	5555542100	4443210000	..	+	++++
13	CNS syphilis	5443210000	4431000000	20	+	++
14	Paresis	5210000000	4432000000	..	+	++++
15	Paresis	5554321000	3444321000	153	+	++++
16	Paresis	4432100000	3432000000	140	+	++++

Bearing in mind that the maximum change in the gold test is represented in the nomenclature by 5 and that the maximum change in the mastic test is represented by 4 and that lesser numerical values differ proportionately, it is evident from a study of Table 1 that the mastic curve on eleven of the sixteen fluids embodied therein pointed to the same clinical condition as did the gold curve. On fluids number 1 and 5, the gold curves were slightly atypically paretic while the mastic curves were typically paretic and the patients were undoubted paretics. On fluid number 2 the mastic curve was slightly atypical while the gold curve was quite typical of paresis. The gold curve of fluid number 8 was characteristic of paresis; the mastic curve, however, was not the typical paretic one. The patient was hemiplegic and presented no definite mental symptoms; possibly he represented one of the so-called preparetic paretics. Fluids number 15 and number 16 were from the same patient, but were taken at different times;

TABLE 2.—The Mastic Reaction on Fluids Having a Positive But Nonparetic Colloidal Gold Reaction

Fluid No.	Diagnosis	Gold	Mastic	Cell Count	Pandy Test	Cerebrospinal Fluid Wassermann Reaction
17		0011132100	0024441000	..	+	
18	CNS syphilis	4333210000	3320000000	84	+	++++
19	Optic atrophy	0112100000	1210000000	46	+	—
20	Tabes	0111000000	2110000000	5	±	++++
21		0001000000	0010000000	..	—	—
22	CNS syphilis	0132100000	2110000000	9	±	++++
23	CNS syphilis	0012100000	0121100000	..	+	
24	Optic atrophy	0112000000	0110000000	38	+	++++
25	Tuberculous meningitis	0012100000	0121100000	63	+	—
26	CNS syphilis	0002331000	0231000000	18	+	—
27	Poliomyelitis	1231000000	1210000000	..	+	—
28	Poliomyelitis	0120000000	2100000000	..	+	—
29	Brain tumor	0012100000	0121000000	3	±	—
30	Tabes	0123410000	2310000000	34	+	++++
31		0012210000	0210000000	..	+	—
32	Tabes	0011000000	0011100000	..	+	
33	Tabes	0011000000	0111000000	..	+	
34	Tabes	2110000000	1332000000	3	±	++
35	Tabes	1112332000	0132100000	..	+	++++
36		0133210000	1431000000	..	—	
37		0001320000	0121000000	..	+	
38		0122310000	0233100000	..	+	++++
39		0001000000	0001000000	88?	+	—
40	CNS syphilis	0121000000	0221000000	128	+	++++
41	CNS syphilis	0121000000	0110000000	..	±	++++
42	Tabes	0001000000	0000000000	1	±	—
43	Tabes	0012110000	2311000000	10	+	++++
44	CNS syphilis	0123100000	0111100000	15	+	++++
45	Tabes	0013321000	1121000000	11	+	++++
46	CNS syphilis	1100000000	1100000000	..	..	—
47		0011000000	0000000000	10	—	—
48	CNS syphilis	1233100000	1210000000	33	+	++++
49	Tabes	0122100000	0110000000	10	+	—
50		0121000000	0000000000	6	—	—
51	Facial palsy	0122100000	1210000000	169	+	—
52	Lethargic encephalitis	0122000000	1110000000	0	+	—
53	Tabes	1243100000	1111000000	20	±	—
54	Optic atrophy	0123100000	1100000000	8	±	—
55	Cord tumor	1122222232	1134443221	9	+	—
56	Tabes	4332100000	4310000000	46	+	++++
57	Posterolateral sclerosis	0111000000	0111000000	..	—	—
58	Uremia	0012210000	4442000000	6	—	—
59	Tabes	2223310000	2221000000	15	+	++++
60	Transverse myelitis	0234110000	1100000000	4	+	—
61	CNS syphilis ?	0123300000	1320000000	3	+	—
62	Cord tumor	1122332100	0012100000	1	+	—
63	Tubercle of cerebellum	0100000000	1100000000	4	+	—
64	CNS syphilis	1224322100	1432000000	28	+	++
65	Paresis	2233433200	4443211000	21	+	++++
66		0011100000	0000000000	12	±	—
67		0012100000	0000000000	3	—	—
68	Transverse myelitis	0012200000	1100000000	5	±	—
69	CNS syphilis	1223321000	2310000000	45	+	++++
70		1122332200	0111112100	65	+	—
71	Optic atrophy	3334421000	3310000000	12	+	—

the patient presented the typical mental changes seen in paresis. It is to be noted that both mastic curves and one of the gold curves were atypically paretic.

From a study of Table 1 it is seen that there is but one mastic curve, that of fluid number 8, which represented a definite deviation in type from its duplicate gold curve, and that in no instance was there a paretic mastic curve with a negative gold curve or vice versa.

Six of the fifty-five fluids reported in Table 2 gave a negative mastic test with a positive gold test. With three of these fluids the change did not amount to at most more than a borderline pathologic change, and with none of the fluids did it reach higher than a number 2 change. Three of the fluids gave a paretic type of mastic curve and a nonparetic, although positive, gold reaction. In fluid number 56 the gold curve was of a borderline paretic type, but although the patient was definitely syphilitic, there were no mental changes. Fluid number 58 presented a difference in type of reaction for which no satisfactory explanation could be found. In fluid number 65 the mastic test, although differing from the gold, represented the actual condition of the patient, who was a well advanced paretic.

TABLE 3.—The Mastic Reaction on Fluids Having a Negative Colloidal Gold Reaction

Fluid No.	Diagnosis	Gold	Mastic	Cell Count	Pandy Test	Cerebrospinal Fluid Wassermann Reaction
72 to 96	Psychoneuroses, psychoses, epilepsy, etc.	0000000000	0000000000	8 or less	—	—
97	Syphilis...........................	0000000000	0100000000	7	—	—
98	Syphilis...........................	0000000000	0000010000	3	—	—
99	"Headache"......................	0000000000	0100000000	3	—	—
100	Tabes..............................	0000000000	1000000000	6	—	—

Table 3 shows that twenty-seven fluids were normal as regards cell count, Pandy test, and Wassermann reaction and gave negative curves with both gold and mastic tests. With four of the fluids giving a negative gold test there was a slight change in one of the tubes of the mastic test. Two of the fluids forming this group were from patients having a positive blood Wassermann test. Another of the fluids was from a patient presenting the clinical picture of tabes and suffering with attacks of abdominal pain. It is obvious, therefore, that of the 100 fluids examined the mastic test was never definitely positive when the gold test was negative. It was negative with five fluids when the gold test was mildly or doubtfully positive (i. e., tests with a maximum change of a number 2 or less). It is to be noted that from the standpoint of a diagnostic or etiologic aid, the mastic reaction is nearly, if not equally, as valuable as the gold test. I feel that those types of change other than that called the paretic type do not permit one to say definitely that a fluid is a syphilitic one, a meningitic one, etc. The so-called paretic curve is diagnostic of paresis only when accompanied by a positive fluid Wassermann reaction. The results of colloidal examinations of the spinal fluid should be considered from a relatively broad aspect and not as a means of pointing conclusively to an anatomic or etiologic diagnosis, or as a criterion to be used for determining the therapeutic value of a method of treatment.

LIMITING FACTORS OF THE MASTIC TEST

The limiting factors have been found to be primarily: the length of time that can elapse between the time the fluid is drawn and the time it will first give a curve differing from that made with the fresh fluid; the length of time that the mastic emulsion can be preserved for use; the effect of admixture of blood; and finally, a point to be considered later in explanation of the discrepant results of certain investigators of this test.

In general it may be said that the results obtained from fluids kept in a sterile manner, at room temperature, for longer than forty-eight hours, are of doubtful value. In certain instances fluids have been found—usually paretic fluids—that will give, after standing a week, a curve exactly similar to that made when fresh.

The use of old mastic emulsions did not appear to be as deleterious as that of old specimens of fluid. The mastic emulsion after standing for some time, gradually lost its opalescency, and it appeared to be wise to use an emulsion made up within the week.

Fluids containing blood, in macroscopic quantities, were found to be as entirely unsuitable for the mastic test as for the gold test.

EXPLANATION OF DISCREPANT RESULTS OF SOME OF THE PREVIOUS INVESTIGATORS

At the beginning of this work various specimens of mastic were examined regarding their suitability for the test. Six lots of mastic were examined, four from local firms and two from out of state firms. Two of these lots of mastic were found to be entirely unsatisfactory for use, giving both quantitatively and qualitatively many false positive curves, as compared with the gold curves of the same fluids. All of the samples of mastic had about the same general appearance. It is evidently only by trial that one may be certain of a suitable supply of mastic. This unsuitability of certain samples of mastic is suggested as a possible source of the discrepant results reported by certain investigators.

CONCLUSIONS

1. The results of the investigators who have made use of the mastic test (Emmanuel-Cutting) have been relatively discordant.

2. The examination of fluids by both the colloidal gold and mastic tests has indicated that the information gained from the mastic test is in close agreement with that obtained from the gold test.

3. Certain samples of gum mastic have been found to be entirely unsuited for use in performing the mastic test. This may serve to explain the conflicting reports of certain workers with this test.[13]

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[13] The recent article by Camp on "Colloidal Mastic Test on Cerebrospinal Fluid" (Am. J. Syphilis **4**:301, 1920), which is in substantial agreement with the conclusions reached herein, came to my attention after this article had been submitted for publication.

THE PHYSIOLOGIC SIGNIFICANCE OF THE BABINSKI TOE RESPONSE *

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The abnormal plantar response that occurs in disease of the pyramidal tract, as described by Babinski,[1] consists of a group of simultaneously occurring movements, the most conspicuous of which are dorsiflexion of the big toe, separation—generally associated with plantar flexion of the other toes and eversion of the foot. The reflexes with which the phenomenon should properly be identified are, therefore, the coordinate or purposive reflexes. These reflexes, in addition to exhibiting themselves in simultaneous contraction of a large number of muscles, are like the Babinski response in that they occur exclusively or are especially prominent after the cerebral influence has been removed. Sherrington [2] and Riddoch [3] give details concerning these reflexes in the "spinal" animal and "spinal" man.

PREVIOUS INTERPRETATIONS OF THE BABINSKI TOE RESPONSE

Among the explanations of the origin and mechanism of the toe response are: Knapp [4] attributed the dorsiflexion of the big toe to a greater permeability of the neuron paths of the extensor longus hallucis. Van Woerkom [5] considered this movement of the big toe a manifestation of a primitive function existing when man, in his evolution, abandoned an arboreal life to lead one of terrestrial progression. Marie and Foix,[6] noting that with extension of the big toe there is a shortening of the stimulated limb, concluded that the reflex represents the flexion phase of the step, or of the "mark-time" movements of Freusberg, Sherrington and others. They thus consider the Babinski toe response to be the "minimal motor response" of that phase of the step in which the limb is advancing to a new position.

Walshe,[7] who studied the flexion reflex, flexor spasms and the Babinski toe reflex, maintains that all these phenomena are identical

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* Read before the Section on Nervous and Mental Diseases at the Seventy-First Annual Session of the American Medical Association, New Orleans, April, 1920.

1. Babinski, M. J.: Compt. rend. Soc. de biol., 1897; Semaine méd. **17**: 321, 1898.
2. Sherrington, C. S.: J. Physiol. **5**:28, 1910.
3. Riddoch, George: Brain **40**:264, 1917.
4. Knapp, P. C.: Rev. Neurol. & Psychiat. **5**:825, 1907.
5. Van Woerkom: Rev. Neurol. **22**:285, 1912.
6. Marie and Foix: Rev. Neurol. **23**:657, 1912.
7. Walshe, F. M. R.: Brain **37**:269, 1915.

in character. The toe response is thus part of that type of flexion reflex which is purely defensive, such as the withdrawal of the limb on the application of a hurtful stimulus. Whereas Marie and Foix look upon the toe response as the "minimal motor response" in the flexion phase of the step, Walshe considers contraction of the hamstring muscle, which occurs synchronously with the toe response, as the "minimal motor response" of the movement of defense.

Any one of these theories is open to the objection that it does not explain the various movements that accompany dorsiflexion of the big toe, such as the fanning out and plantar flexion of the other toes, eversion of the foot and especially contraction of the quadriceps cruris, an extensor of the leg, and contraction of the gluteus maximus, a pure extensor of the thigh. That there is synchronously with the Babinski toe response contraction of the quadriceps cruris has been noted by Cohn,[8] Jacobsohn and Caro,[9] Erwin Loewy[10] and myself.

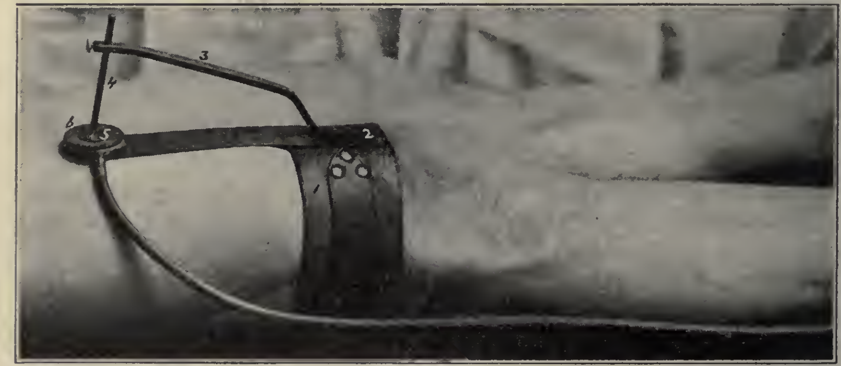

Fig. 1.—Apparatus for recording the movements in the segments of the limbs

But I have also noted that there is simultaneously an extension of the thigh on the hip. The contraction of the gluteus maximus producing this movement can be frequently observed when the toe sign is obtained while the subject is in the prone position. This is shown graphically in Figures 4 and 5.

To obtain these graphic records, I employed this method: A piece of cord attached to the big toe passed under a pulley to a writing lever in contact with the drum of a kymograph. Dorsiflexion of the big toe was thus recorded by an upstroke of the lever. To record the other movements in the limb I employed the apparatus shown in Figure 1.

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[8] Cohn, T.: Neurol. Centralbl. **30**:1107, 1911.

[9] Jacobsohn and Caro: Neurol. Centralbl. **31**:420, 1912.

[10] Loewy, Erwin: Neurol. Centralbl. **32**:90, 1913.

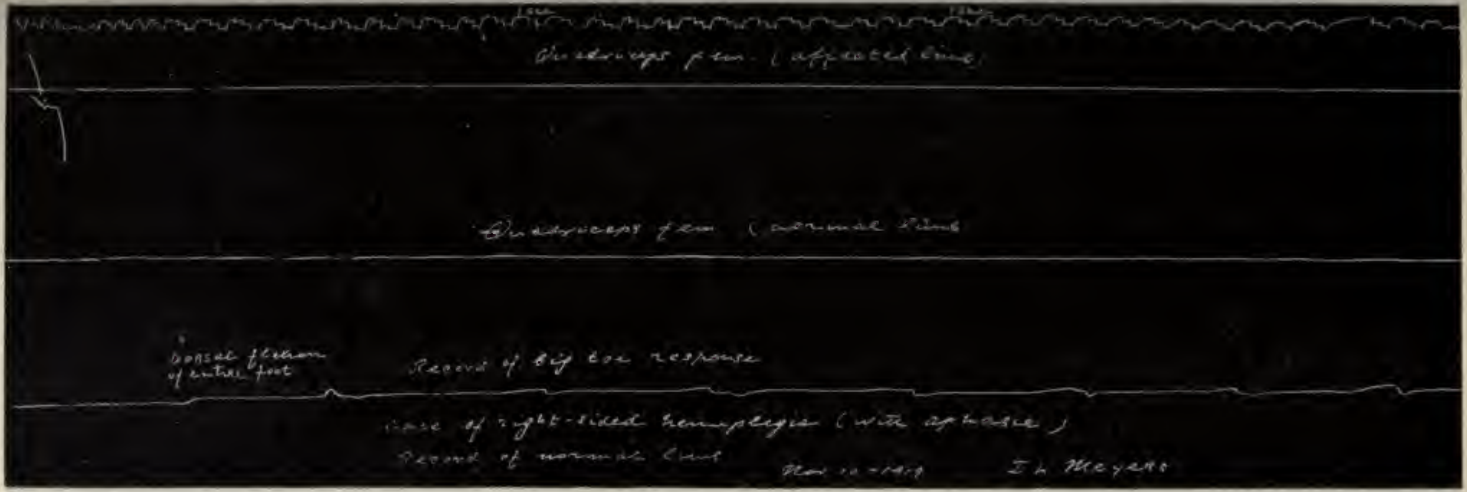

Fig. 2.—Record of the normal plantar response.

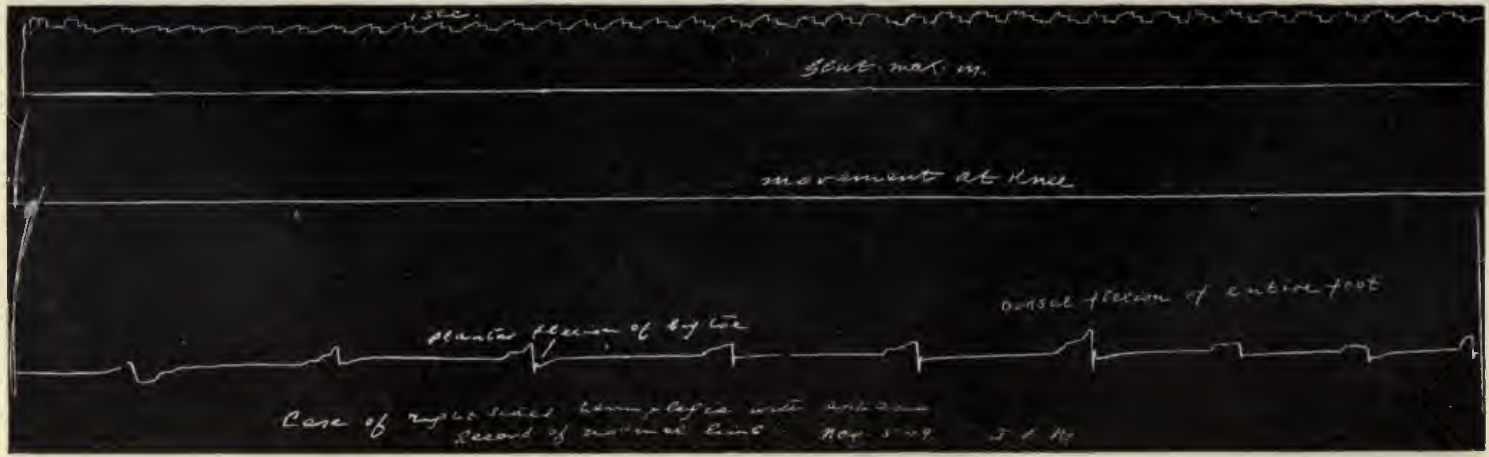

Fig. 3.—Record of the normal plantar response.

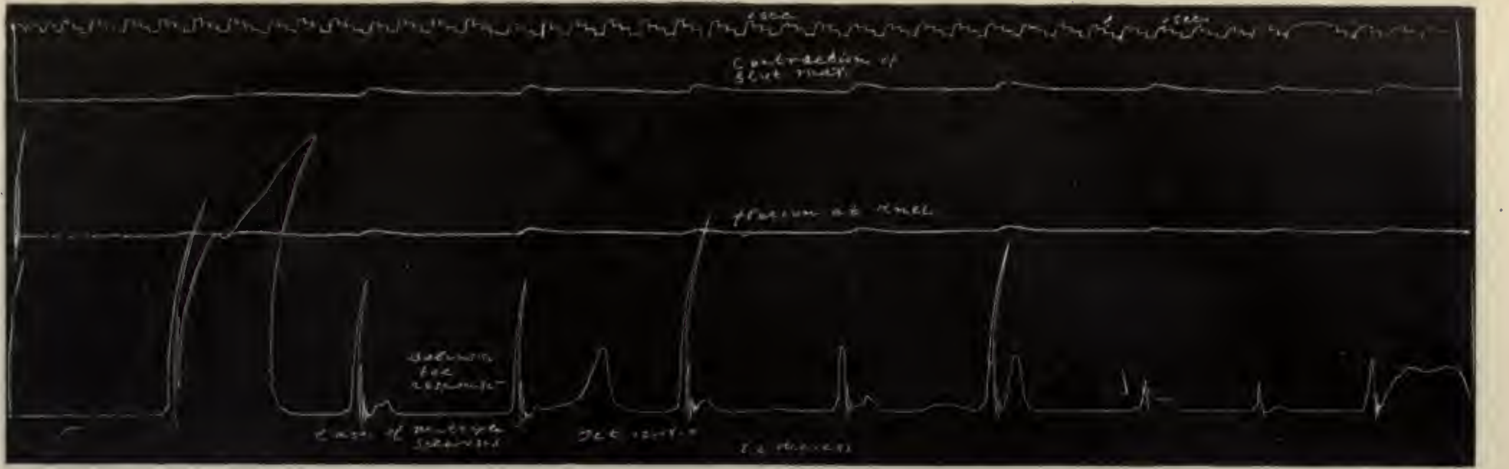

Fig. 4.—The Babinski toe response with the accompanying movements in the other segments of the limb.

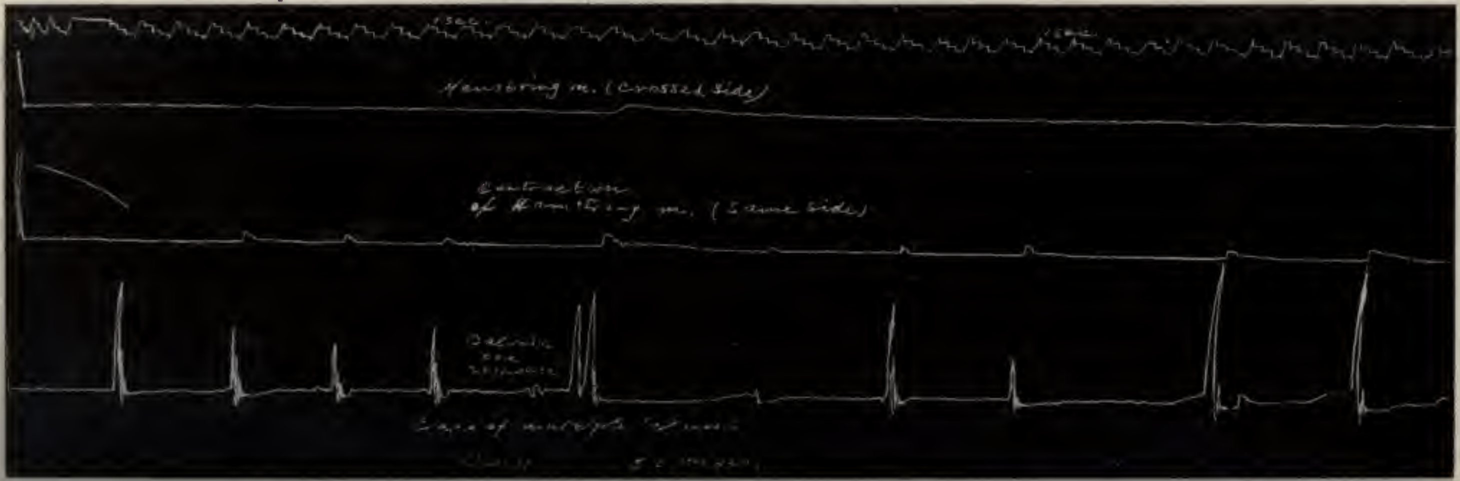

Fig. 5.—The Babinski toe response with the accompanying movements in the other segments of the limb.

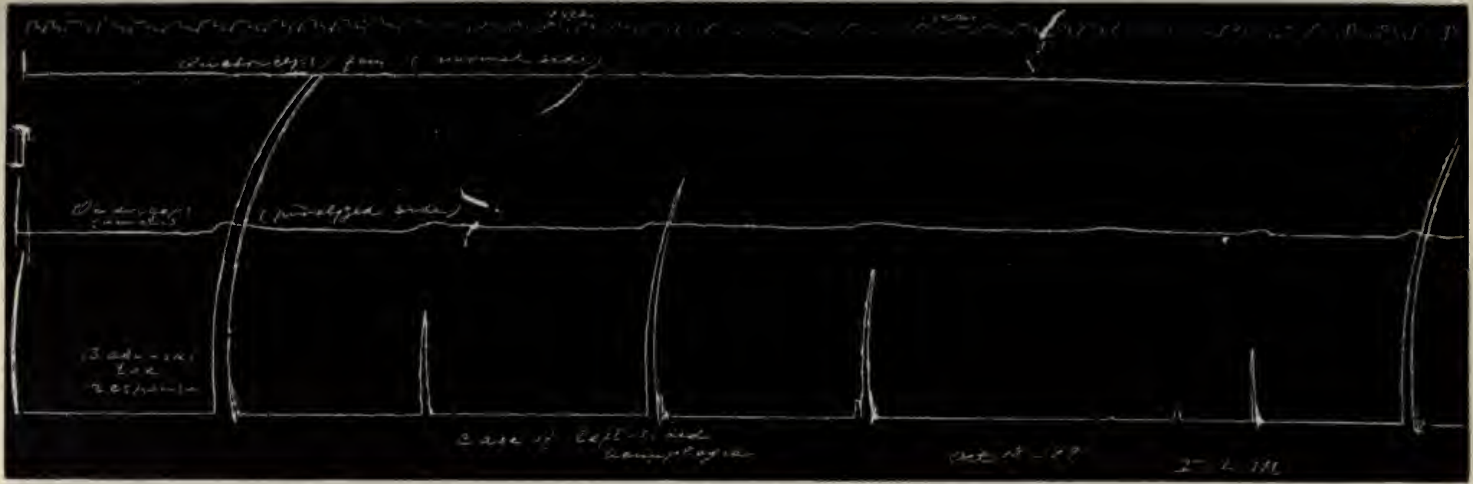

Fig. 6.—The Babinski toe response with the accompanying movements in the other segments of the limb.

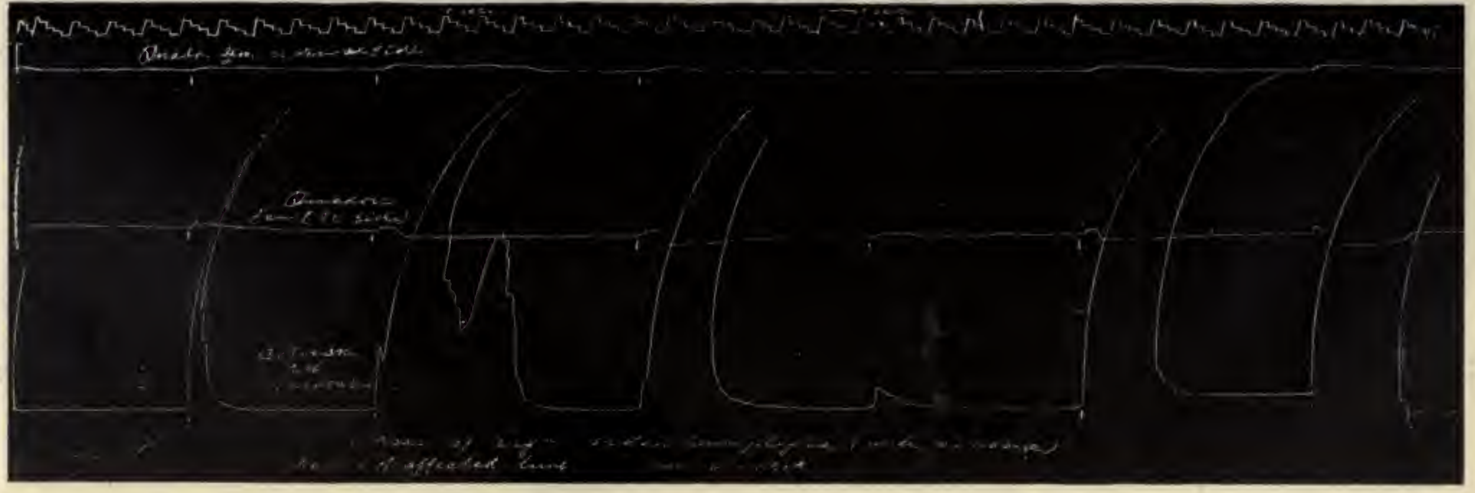

Fig. 7.—The Babinski toe response with the accompanying movements in the other segments of the limb.

To the middle of a leather braclet (1) is fastened a metal plate (2). To this plate are riveted a semirigid metal strap over 5 inches long (7) which extends along the surface of the limb in its long axis and a rigid bar (3) rising from its point of attachment so that its end is a couple of inches above the free end of the metal strap. Through the free end of the bar passes a metal rod (4) which rests by a small circular plate (5) on an elastic air chamber (6), fastened to the free end of the metal strap. The rod is secured by a thumb screw where it passes through the bar so that the circular plate is kept firmly in contact with the air chamber. By a rubber tube the air chamber communicates with a Marey's tambour and a kymograph.

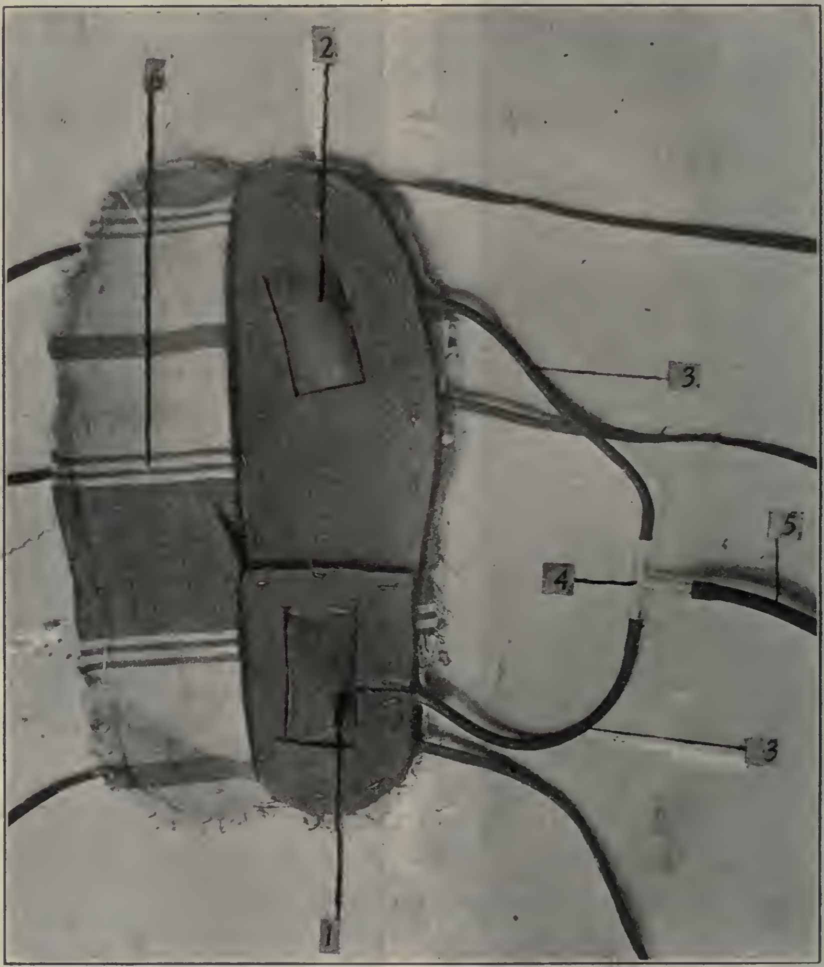

Fig. 8.—Apparatus for recording the movements of the foot in walking.

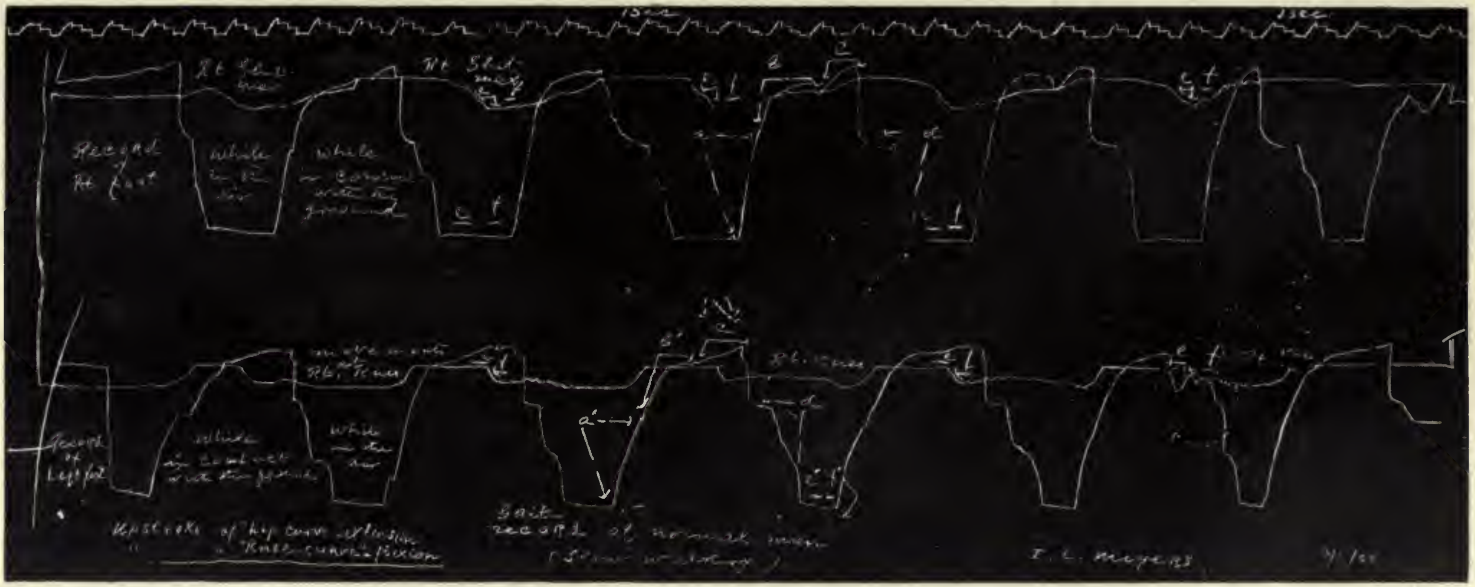

Fig. 9.—Record of the various phases of the step.

To record the contraction of the quadriceps cruris and the hamstring muscle, the bracelet encircled the thigh at the lower end and the air chamber rested, respectively, at about the middle of the quadriceps or on the hamstring muscles just below the gluteal fold. To record the contraction of the gluteus maximus the bracelet encircled the thigh at about its middle and the air chamber rested on the lower part of the muscle which extends between its origin at the sacrum and coccyx and its insertion. I recorded the flexion of the knee on the thigh by having the bracelet encircle the thigh at about its middle and the air chamber rest at the lower end of the popliteal space between the two heads of the gastrocnemius. I have graphic records of the movements in the limbs associated with the Babinski toe response from fourteen hemiplegic or paraplegic patients. The results obtained are shown in Figures 4 to 7. The records show that simultaneously with the Babinski toe response there was invariably a contraction of the homolateral quadriceps cruris, gluteus maximus and hamstring muscles. This response was not accompanied by movement in the contralateral thigh (with the exception of slight contraction of the hamstring muscles) when the stimulus was mild. When, however, the stimulus was strong, the contraction of the quadriceps was immediately followed by its relaxation and this was accompanied by contraction of the contralateral quadriceps. In the one case of Friedreich's ataxia I have obtained results in some respects, directly the reverse of the preceding. The records showed that simultaneously with the Babinski toe response there was flexion of the knee with relaxation of the homolateral quadriceps and gluteus maximus (Figs. 11 and 12). This may have been due to the muscular atonia in this disease. In contrast to the response on the paretic side in hemiplegics, was that obtained from the normal limb. When the stimulus was strong the limb underwent a rapid movement of withdrawal, entirely too rapid to be properly recorded; when the stimulus was mild, there was dorsiflexion of the foot, generally immediately followed by plantar flexion of the big toe (the only toe the movements of which were recorded), with no movement in the other segments of the limb (Figs. 2 and 3).

MOVEMENTS OF THE LIMBS IN LOCOMOTION

Since locomotion is the primary and essential function of the lower limbs, I wished to determine whether this combination of movements occurring synchronously with the Babinski sign is reproduced by the limbs at any time during walking. First I considered that the inner metatarsal, as is shown by a number of anatomic facts,[11] forms the

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[11] Frazer, J. Ernest: Anatomy of the Human Skeleton, London, J. A. Churchill, 1914.

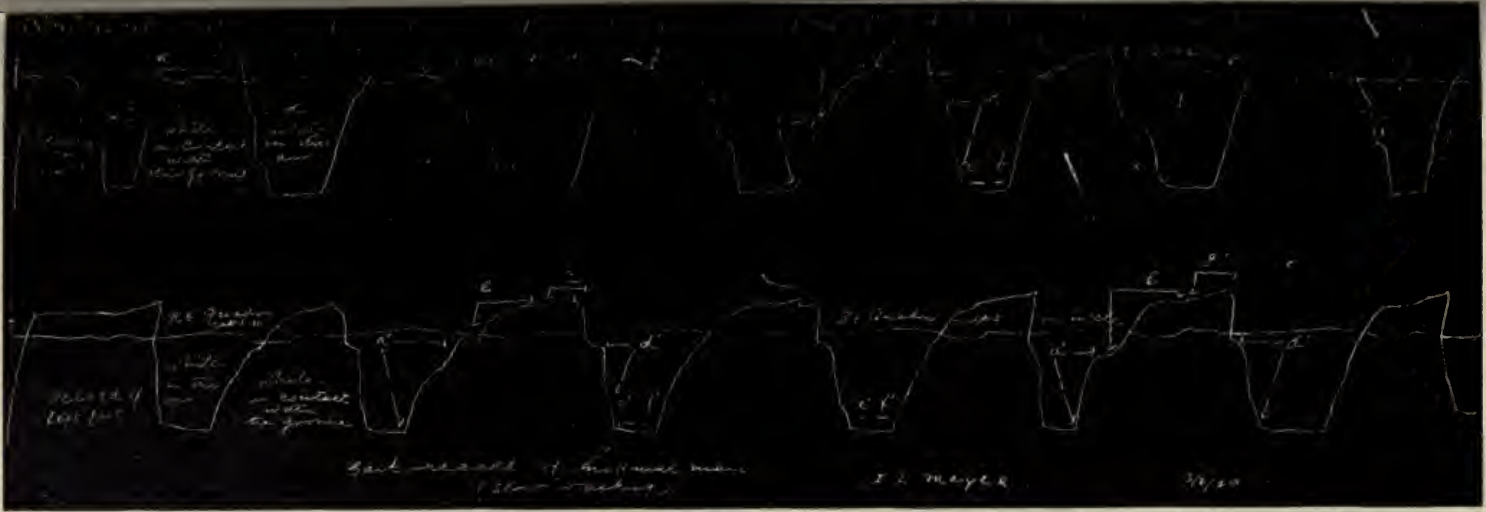

Fig. 10.—Record of the various phases of the step.

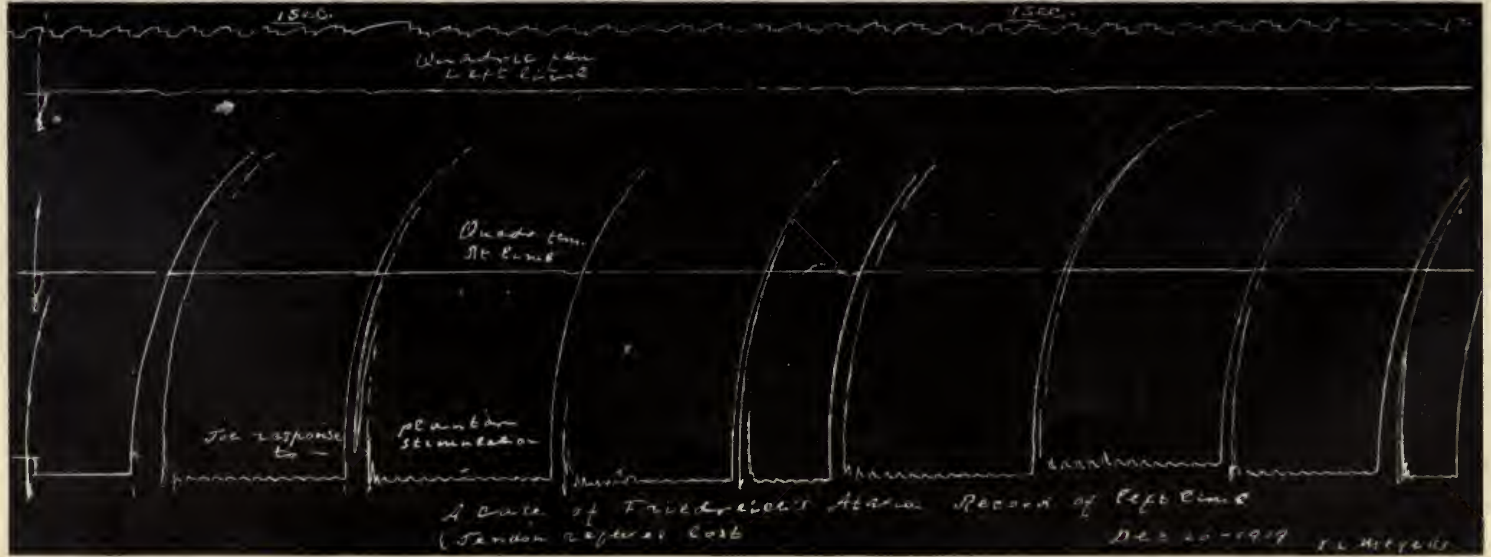

Fig. 11.—The Babinski toe response in a case of Friedreich's ataxia.

medium through which pressure is exerted on the ground during the propulsion period of the step. The outer metatarsus, with its practically immobile articulations, serves chiefly as a guide and point of support for the body and limb while they are rotating anteriorly during this period of the step. Accordingly I fastened to each foot of a normal person a pneumatic pad, such as I employed in my experiments on the locomotor disturbances in disease of the cerebellum (Fig. 8). The anterior air chamber was placed under the inner metatarsal head instead of the center of the ball of the foot as in my previous studies. Records were thus obtained of the movements of the foot while in contact with the ground, as well as of the length of time it is in the air. Movements of the other segments of the limbs during each phase of the step were recorded at the same time by means of the apparatus employed for recording the movements that accompany the Babinski sign. The records obtained are shown in Figures 9 and 10. They show: (1) *a*, the upstroke which initiates the high curve of the record. This corresponds to the moment when the foot is deposited on the ground, coming in contact with it by the heel (Pettigrew[13]). The resulting compression of the air bulb at the heel causes the lever to become elevated. The associated movements in the other segment of the limbs during this phase of the step are: The hip is slightly extending, the knee is slightly flexing, the quadriceps cruris is contracting and the quadriceps of the contralateral limb, which is at this moment exerting pressure on the ground by the toes, (phase *d′*) is relaxing. (2) Immediately following *a*, phase *b* sets in, which is but a continuation of *a*. During this phase (*b*) the whole foot comes in contact with the ground, especially at the heel. The associated movements in the other segments of the limb during this phase are the same as in *a*, except that the extension at the hip is here greatly intensified. The position of the limb is now to a minor degree, similar to that assumed by the limbs at the beginning of a jump, when in order to exert great and rapid pressure on the ground, they are considerably flexed at the knees. The contraction of the quadriceps during these phases, while the knee is slightly flexing (action of the hamstring muscles) is in accord with the view of Winslow,[14] Hunter,[14] Duchenne[15] and

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[12] Meyers, I. Leon: Locomotor Disturbances in Disease of the Cerebellum, Arch. Neurol. & Psychiat. **2**:376 (Oct.) 1919.

[13] Pettigrew, J. Bell: Animal Locomotion, New York, Appleton & Co., 1874, pp. 8 and 9, 56-59; Design in Nature, New York, Longmans, Green & Co. **3**:1077 and 1122, 1908.

[14] Winslow, J. B.: Quoted by C. E. Beevor: The Croonian Lectures, London, Adlard and Son, 1904, p. 52.

[15] Duchenne, G. B.: Physiologie des Mouvements, 1867, p. 766.

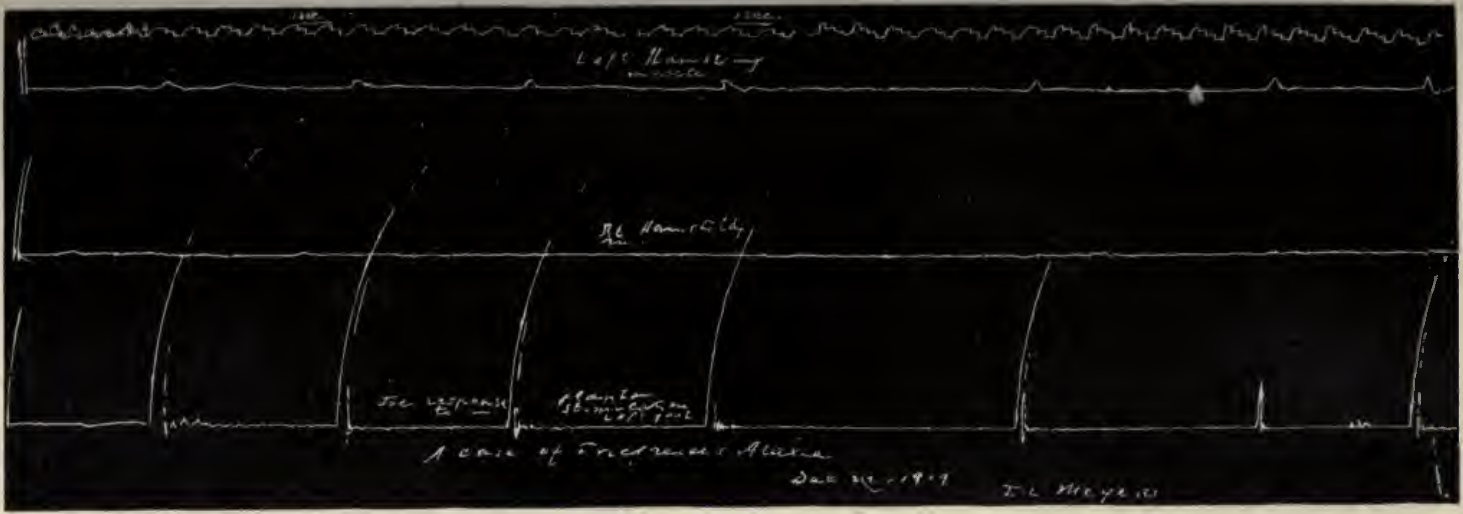

Fig. 12.—The Babinski toe response in a case of Friedreich's ataxia.

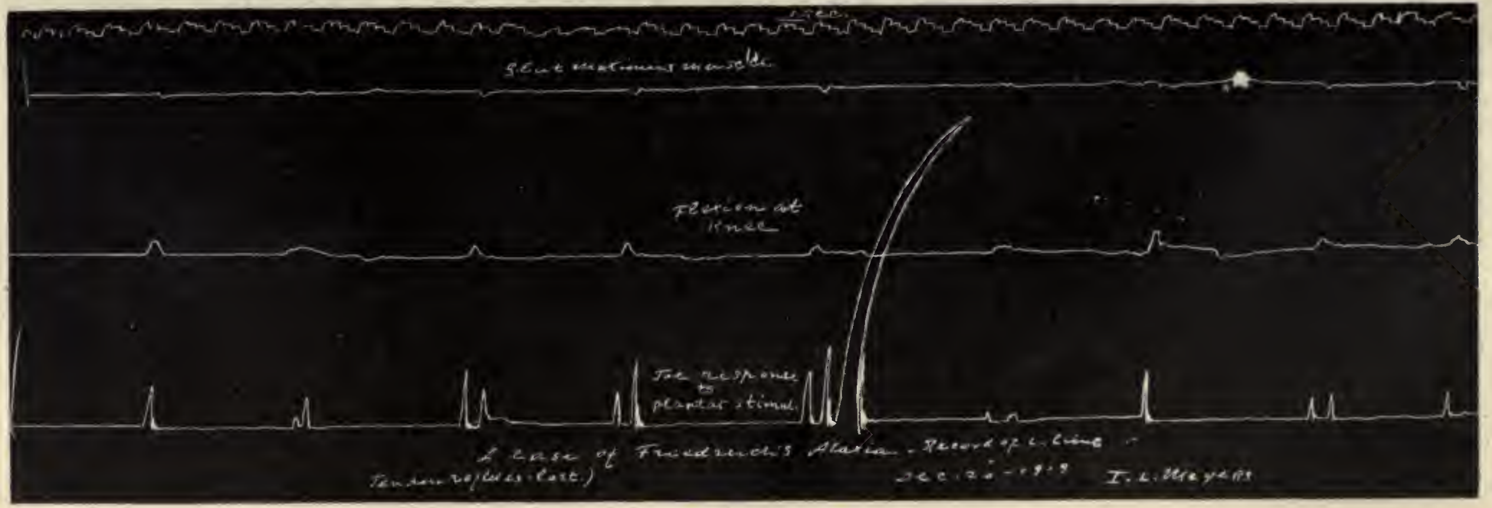

Fig. 13.—The Babinski toe response in a case of Friedreich's ataxia.

others that in movements that are to be accurate and precise, the antagonists act simultaneously with the protagonists. (3) Phase *c*, the pressure on the ground, already at play in the preceding phase, becomes intensified, being now, according to the estimate by Carlet,[16] one-fifth in excess of the body weight. The pressure is now at the ball of the foot exclusively, and in my experiments, at the sesamoid bones of the inner metatarsal, where the air-bulb for the ball of the foot was placed. The associated movements in the other segments of the limbs during this phase are: The homolateral hip is markedly extending, the knee is also markedly extending, the quadriceps cruris is contracting and the quadriceps of the contralateral limb, which is at this time returning to the ground (phase *f′*), is also contracting. (4) Phase *d*, which represents the period when the limb exerts pressure on the ground by the toes only. The pressure on the ground at the toes causes some compression of the air-bulb at the ball of the foot, and is thus recorded by a partial elevation in the curve of the record. During this phase the homolateral hip is markedly extended, the knee is markedly flexed, the quadriceps is relaxed and the quadriceps in the contralateral limb, at this time in phases *a′* and *b′*, is contracted. *d* terminates the period during which the foot is in contact with the ground, and is followed by (5) *e*, which corresponds to the time during which the foot is in the air and advancing. The movements in the other segments of the limbs during phase *e* are: The hip is flexing, the knee continues to be in flexion, the quadriceps is relaxing and the quadriceps of the contralateral limb, at this time in phase *b′*, is contracting. (6) *e* is followed by phase *f*, when the limb is returning to the ground. During this phase the homolateral hip is slightly extending, the knee is extending, the quadriceps is contracting and the quadriceps of the other limb, at this time in phase *c′*, and exerting pressure on the ground by the ball of the foot, is also contracting. It will be seen thus that a complete step is composed of six different and distinct phases, four of these phases forming the propulsion period of the step and two more the period when the foot is in the air.

On looking now at the phases of the step for the combination of movements, which is observed to occur synchronously with the Babinski sign, we find that it is reproduced in phases *a* and *b*, when the body is exerting pressure on the ground mainly at the heel. During this phase of the step, the following movements take place: The big toe becomes dorsiflexed on the foot; the other toes, as shown by Pettigrew,[13] spread out and generally undergo plantar flexion; the whole foot from the direction of movement of the body and limb, which is not only forward, but as shown by Pettigrew and Morey, also horizontally outward, becomes everted; the knee is slightly flexed; the quadriceps cruris and the gluteus maximus are in contraction, and the quadriceps of the contralateral limb, at this time exerting pressure on the ground by the toes (phase *d'*), is relaxed.

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[16] Carlet, G.: Ann. d. sc. naturelles **16**:5, 1872.

THE PHYSIOLOGIC SIGNIFICANCE OF THE BABINSKI TOE RESPONSE

The following theory thus suggests itself. It may be assumed (Marie and Foix) that the function of progression is represented in the spinal cord (l' automotisme medullaire); that normally the movements of progression are kept more or less in check by inhibitory influences of the cerebrum. When, however, inhibition from the cerebrum is cut off as a result of disease of the corticospinal tracts, the spinal centers of progression reassert themselves whenever a stimulus reaches them from the limbs, and the stimulated limb or both limbs execute the stepping reflex in a minor degree. The stepping reflex exhibits itself in the Babinski toe response which represents phase *b*, the first and most important phase in the propulsion period of the step. Phase *b* is the only manifestation of the reflex when the stimulus is mild. When the stimulus is intense, the stepping reflex reaches a much wider extent and passes through, to a minor degree, also phase *d*. The quadriceps cruris of this limb, in contraction in phase *b*, thus becomes relaxed, and there is usually at the same time a movement of plantar flexion of the big toe following the original dorsiflexion. A pronounced reflex of this type affects also the contralateral limb, and, while the stimulated limb is in phase *d*, the quadriceps of the former limb contracts. The most striking features of the Babinski toe response are, the dorsiflexion of the big toe, the spreading out of the lesser toes and the eversion of the foot. This is probably due to the close proximity of the muscles involved in these movements to the point of application of the stimulus (the plantar surface of the foot). If, however, the reflexogenous zone (of the Babinski toe response) extends over the entire limb, (and occasionally also the lower part of the abdomen) the stimulus when applied to the upper part of the limb may, as noted by Marie and Foix, produce extension of the limb (marked action of the gluteus maximus and the quadriceps cruris) and no dorsiflexion of the big toe. The effect of the stimulus is in this instance, too, produced chiefly on the muscles close to the point of stimulus and not on the parts distal to it.

This theory of the Babinski sign explains also the so-called crossed plantar response, frequently observed in paraplegia and occasionally in hemiplegia, which consists of plantar flexion of all the toes with extension of the foot. The phenomenon has been studied and interpreted in various ways by Byron Bramwell,[17] Knapp,[4] Fairbanks[18] and others. If the Babinski toe response represents the propelling period of the step that occurs in phase *b*, the contralateral limb at this time being in phase *d'*, the effect of the stimulus to one limb, when the irritability of the nervous system is so great that the reflex affects the other limb also, must be that of plantar flexion of all the toes in the latter limb, the foot being at the same time fully extended at the ankle. Frequent absence of the crossed plantar response may be accounted for by the remoteness of the toes in the nonstimulated limb from the point of application of the stimulus.

This theory may also explain why the Babinski toe response, while obtainable in some conditions by a stimulus applied to any part of the limb and even the lower part of the abdomen, is obtained with such marked facility by pressure at certain foci in the foot and leg. These "receptive foci" are, I believe, the heel, the ball of the foot and the area under the outer malleolus. It will be noted that pressure at the heel and the ball of the foot reproduces the condition which is associated with dorsiflexion of the big toe in walking, and that pressure under the outer malleolus produces an effect on the peroneus longus, the existence of which is, as stated by Huxley,[19] absolutely distinctive of the human foot, a muscle that is essentially an organ of locomotion.

25 East Washington Street.

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[17] Bramwell, Byron: Rev. Neurol. & Psychiat. **1**:795, 1903; ibid. **9**:49, 1911.
[18] Fairbanks, A. W.: Rev. Neurol. & Psychiat. **9**:225, 1911.
[19] Huxley, T. H.: Man's Place in Nature, New York. O. Appleton & Co., 1896, p. 124.

THE MOTOFACIENT AND NONMOTOFACIENT CYCLES IN ELEVATION OF THE HUMERUS *

BYRON STOOKEY, A.M., M.D.

NEW YORK

The generally accepted view that in raising the humerus to a straight angle, the deltoid elevates the arm approximately to a right angle, after which elevation is completed by scapular rotation, is not supported by our investigation. The deltoid alone is unable to raise the humerus beyond 60 degrees while elevation from this to approximately 115 degrees involves scapular rotation, after which the deltoid again becomes active and completes the elevation.

Inspection and palpation or electrical stimulation of the deltoid will verify the active rôle it plays in elevation of the arm from 115 degrees to a straight angle. The deltoid acts first as an abductor, abducting the arm approximately 60 degrees, and then after elevation to 115 degrees it becomes an adductor, thereby raising the arm through the final arc to 180 degrees.

The origin and insertion of the deltoid are so placed that it may serve this dual function, providing the arm is considered in its position in different stages of elevation. Elevation of the arm has been difficult to interpret, since any movement involves the complex musculature of the shoulder girdle, whose component muscles have wide and diverse origins, and are capable of independent contraction in different parts of the same muscle, subserving at different times different functions.

In paralyses in which the scapula cannot participate in elevation, the deltoid alone is unable to bring the humerus above 60 or 65 degrees. Normally in any abduction beyond 6 degrees, rotation of the scapula takes place and may be made apparent, by fixing the inferior angle of the scapula between the fingers during the act of abduction, when it will be noted that as soon as the humerus reaches an angle of approximately 60 degrees rotation of the scapula occurs. When the humerus is abducted to 75 degrees, the scapula has rotated 20 degrees—thus of 75 degrees abduction, 20 degrees are the result of scapular rotation and only 55 are due to humerus abduction from the scapula. This may be seen by comparing the accompanying illustrations [1] showing the normal relation of the scapula to the humerus

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* Read before the New York Neurological Society, May, 1920.

[1] I wish to express my thanks to Dr. Scholz who kindly took the roentgenograms from which these tracings were made.

and to a perpendicular through the shoulder joint. These tracings were made from a series of roentgenograms taken of the writer under constant conditions. The tube was 42 cm. from the plate, the body erect, and the scapula unsupported. (In lying down, the scapula may receive support due to the weight of the body, etc., and its movements be somewhat altered.)

The arc of elevation should be broken into its functional units rather than into the mathematical components of a right angle and a straight angle. These are not functional units and do not admit of interpreting the relation of scapular rotation in the different cycles of elevation of the humerus. The roentgen ray was used by Steinhausen [2] in studying the movements of the scapula, but he divided the movements into various stages from a mathematical standpoint, rather than from physiologic and anatomic considerations, and thereby failed to realize the proportionate relation of scapular rotation to elevation of the shoulder.

THREE PHYSIOLOGIC CYCLES IN ELEVATION OF THE ARM

We have found the functional units in the elevation of the humerus to be: first, elevation of the arm in which there is little or no rotation of the scapula (60 degrees); second, elevation during which the major portion of scapular rotation takes place (60 to 115 degrees); third, the final arc of elevation in which scapular rotation has relatively ceased and elevation of the humerus is continued to a straight angle (115 to 180 degrees).

First Stage: From a study of Figure 1, it will be noted that the humerus is abducted 60 or 65 degrees, and the scapula rotated 5 or 6 degrees. Of this movement, approximately 55 degrees are due to the action of the deltoid and supraspinatus.

Second Stage: In further abduction and elevation from 60 to 115 degrees, the scapula is rotated an additional 35 degrees. Thus, in this arc of 55 degrees, 35 degrees is accomplished by scapular action and only 20 degrees by the deltoid.

Third Stage: The elevation from 115 to 180 degrees is accompanied by only 10 degrees of scapular rotation. In this arc of 65 degrees the scapula accomplished but 10 degrees, the remainder being unaccounted for by the older view, which attributes all of this final arc, as well as part of the second, to rotation of the scapula.

First Cycle.—During the first stage, the arm is abducted to 60 degrees by the deltoid and also to a lesser extent by the supraspinatus, and is accompanied by a slight rotation of the scapula, more particu-

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[2] Steinhausen: Beitrage zur Lehre von dem Mechanismus der Bewegungen des Schultergürtels, Arch. f. Anat. u. Physiol. 1899, p. 403.

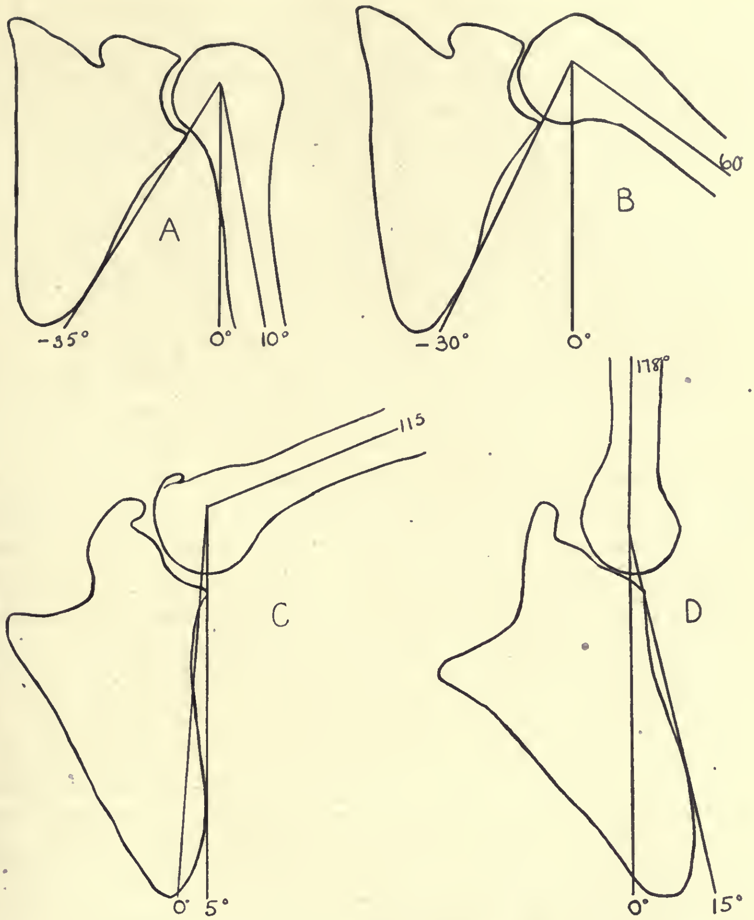

Fig. 1.—Tracings from roentgenograms showing the relation of the scapula to the humerus and to a perpendicular through the shoulder joint in various phases of elevation of the humerus. A line has been carried in each along the axillary border and through the inferior angle of the glenoid cavity. The line through the humerus is carried through the center of its longitudinal axis. *A,* the normal relation of the scapula to the humerus. The scapula forms an angle approximately 35 degrees with a perpendicular, and the humerus 10 degrees. The humerus may be brought to the perpendicular without any alteration in the relative position of the scapula. *B,* the humerus has been raised 60 degrees, during which the scapula has rotated approximately 5 degrees. This rotation of the scapula is rather in the sense of fixation than a participation in elevation of the humerus. *C,* the humerus has been raised to 115 degrees, during which the scapula has rotated 35 degrees from its position in *B,* and 40 degrees from its position in *A.* In elevation from 60 to 115 degrees, the scapula has rotated 35 degrees or 70 per cent. of its total rotation. *D,* the humerus has been raised practically to a straight angle, during which the scapula has rotated an additional 10 degrees from its position in *C,* or a total of 50 degrees from its original position in *A.* In this last cycle of elevation, the scapula has rotated 20 per cent. of its total.

larly in the sense of fixation. Duchenne[3] has accurately pointed out that the rhomboideii and trapezius act as fixators of the scapula, permitting it to serve as a fixed point for the attachment of the deltoid. The supraspinatus serves to help hold the head of the humerus against the glenoid cavity. However, the only action he attributed to the deltoid was abduction of the arm to 90 or 100 degrees. Thus, in the first cycle of elevation, the deltoid and supraspinatus may be said to to the motofacient factors; the rhomboideii, trapezius and serratus magnus serve as nonmotofacient and fixators of the scapula. In this arc, the scapula rotates 10 per cent. of its total rotation and accomplishes but 8 per cent. of the total elevation of the humerus.

Second Cycle.—During the second cycle the scapula subtends an arc of 35 degrees, or 70 per cent. of its total rotation, and 65 per cent. of the elevation of the arm from 60 to 115 degrees. This is the arc of its major rotation. Rotation of the scapula is accomplished by contraction of the serratus magnus, particularly the lower digitations which rotate the inferior angle laterad. This action is also furthered by contraction of the middle portion of the trapezius; the inferior and superior portions, as well as the rhomboideii, serve as fixators, and direct the axis of scapular rotation. During this cycle, the deltoid and supraspinatus act principally as fixators and determine the angle of the humerus with the body. However, they also elevate the arm approximately 20 degrees, yet may be said to be essentially nonmotofacient. In the latter function, they are assisted by the tres major, pectoralis major and latissimus dorsi, which help to hold the humerus against the glenoid cavity as did the supraspinatus in the first stage, and also help in determining the angle which the humerus forms in the horizontal plane with the body.

Third Cycle.—In the third cycle, from 115 to 180 degrees, the scapula participates but 10 degrees, or the scapula rotates 20 per cent. of its total rotation and accomplishes 15 per cent. of the total elevation in this stage. Thus 55 degrees of elevation are accomplished without the assistance of the scapula. This last cycle is performed principally by the deltoid and supraspinatus, more specially the clavicular head of the deltoid. However, this depends on the horizontal angle which the humerus forms with the trunk. The more ventrad the humerus, the more stress is thrown on the clavicular head of the deltoid, and the further dorsad the humerus, the more the median fibers participate. The posterior fibers serve as guy ropes, together with the teres major and latissimus dorsi, to determine the angle with which elevation is accomplished.

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[3] Duchenne: Physiologie des Movements, 1867, J. B. Balliere et Fils.

With the arm at 115 degrees, the supraspinatus and deltoid are ideally placed to exert considerable force in the direction of elevation. It might be said that the supraspinatus muscle and the deltoid now complete the elevation by adduction. Thus the deltoid has again become motofacient, and the scapular muscles essentially nonmotofacient, serving primarily to fix the scapula, though they participate slightly in elevation.

In raising the humerus from zero to 180 degrees, the scapula rotates a total of 50 degrees; the remaining 130 degrees of elevation are humerus action, due chiefly to the supraspinatus and deltoid, more particularly the clavicular head of the latter, especially in the final cycle of elevation.

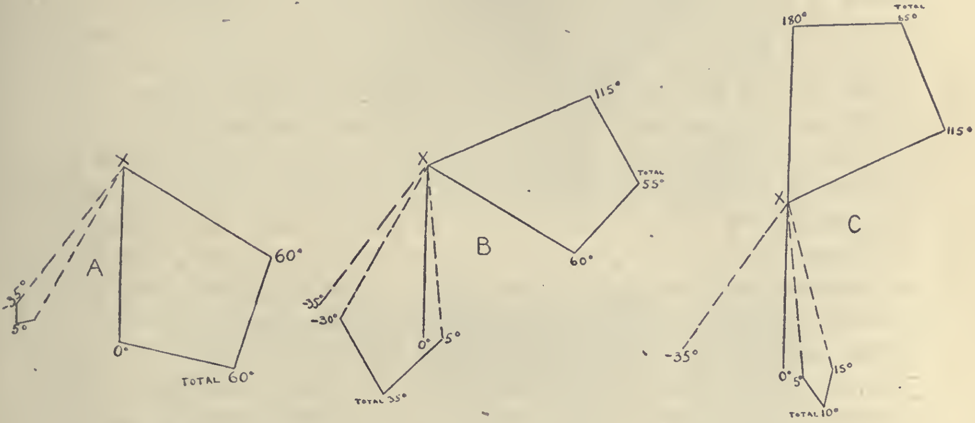

Fig. 2.—Diagrams taken from Figure 1 to illustrate the relationship of rotation of the scapula to elevation of the humerus. *A*, during elevation of the humerus to 60 degrees, the scapula has rotated 5 degrees; 55 degrees represents elevation of the humerus alone. Thus 8 per cent. of elevation is due to scapular rotation, and the scapula rotates 10 per cent. of its total. *B*, elevation of the humerus from 60 to 115 degrees, during which the scapula has rotated but 35 degrees; 20 degrees is the elevation of the humerus alone. The scapula rotates in this cycle 70 per cent. of its total. *C*, elevation of the humerus 65 degrees from 115 to 180, during which the scapula has rotated 10 degrees; 55 degrees is the elevation of the humerus alone and unaccounted for by any rotation of the scapula. In this last cycle of elevation; 15 per cent. is due to scapular rotation. The scapula rotates 20 per cent. of its total.

Thus alternating cycles form the functional units in the different stages of elevation, in the first of which the deltoid is motofacient and the scapular muscles are nonmotofacient; in the second, the scapular muscles are motofacient, and the deltoid, supraspinatus, teres major, pectoralis major and latissimus dorsi are nonmotofacient; while in the third cycle, the deltoid and supraspinatus again become motofacient and complete the act of elevation.

Action of Clavicular Head of Pectoralis Major and Coracobrachialis in Final Stage of Elevation.—Two muscles, whose action in elevation has been ignored, may serve as supplementary factors of no small importance in accomplishing the final stage of elevation. They are the clavicular head of the pectoralis major and the coracobrachialis. These muscles are brought into play particularly when great force in elevation is demanded, or when there is some impairment of the function of the deltoid, or of the muscles which fix and rotate the scapula. The accompanying illustrations taken from a case in which the trapezius was totally paralyzed, due to the severance of the spinal accessory nerve a short distance below its exit, illustrate the action of these two muscles, particularly the clavicular head of the pectoralis major. So far as we have been able to detect, there is total paralysis of all parts of the trapezius in spite of its supply from the third and fourth cervical nerves. This patient has learned to compensate in part for the lost action of the trapezius in elevation of the arm by marked hypertrophy of the clavicular head of the pectoralis major and the coracobrachialis. The former muscle particularly takes an active part in elevation of the arm beyond 115 degrees. When contrasted with the normal side, the clavicular portion of the pectoralis major stands out almost as a separate muscle and may be picked up between the fingers, being approximately 3 to 4 cm. thick. During elevation above 115 degrees the clavicular portion shows definite visible contraction accompanied by outward rotation of the humerus, in which position elevation is more readily accomplished. A front and back view, as illustrated in Figure 3, show the contraction of this muscle in the act of elevation, thus indicating its active rôle in the accomplishment of this movement.

Normally, this function of the clavicular head of the pectoralis major may be brought out by forcible resistance to elevation or by sudden attempts to lower the arm from elevation, particularly if the patient is able to oppose suddenly and vigorously each of these movements. The clavicular portion of the pectoralis major will be seen to take active part in such forcible elevation above 115 degrees, or oppose efforts to lower the arm to 115 degrees.

In this connection, it is interesting to note that phylogenetically the clavicular head of the pectoralis major is present only as such in the higher forms, particularly in the chimpanzee and the gibbon, while in all other primates as well as the lower mammals, it is wanting. In them, the pectoralis major arises from the manubrium and does not reach to the clavicle. However, in those animals in which there is no clavicular head of the pectoralis major, the clavicular portion of the deltoid extends mesially on the clavicle up to the origin of the sternocleidomastoideus. The clavicular portion of the pectoralis major may be considered as a migration of the innermost muscular fibers of the

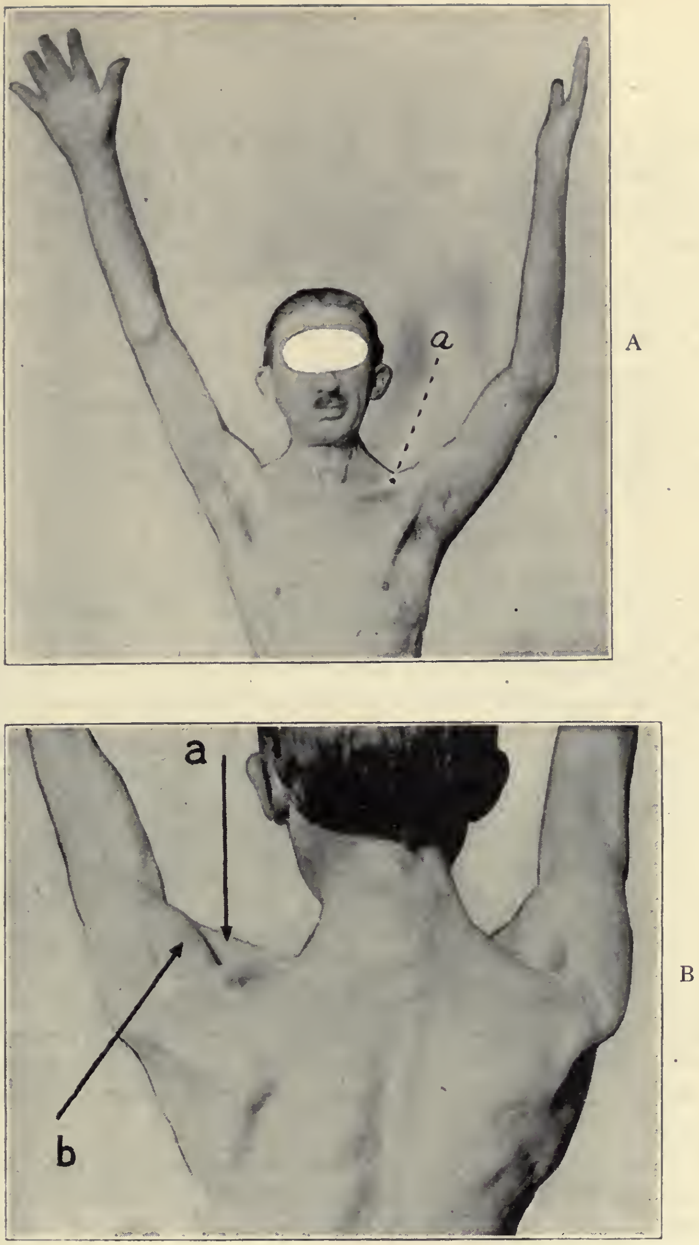

Fig. 3.—Photographs of patient showing marked hypertrophy of the clavicular head of the pectoralis major. The trapezius has been totally paralyzed for ten years. *A,* on the left side, the clavicular portion of the pectoralis major shows marked hypertrophy and stands out almost as a separate muscle. An interval can be seen between the clavicular head and the sternal portion. In movements with the arm at the side, the pectoralis major contracts as a whole and shows no anomaly. The separation of the clavicular and sternal portions takes place only in elevation of the arm above 115 degrees. Note that the arm is held in external rotation. *B,* Posterior view of the same patient showing the active rôle of the clavicular portion of the pectoralis major in elevation of the arm from above 115 degrees. Note also on both sides the contraction of the deltoid, particularly the clavicular head, showing the active participation of this muscle in the elevation of the humerus—a function generally ignored. *a,* clavicular head, pectoralis major; *b,* clavicular head of the deltoid.

clavicular head of the deltoid. This view is perhaps further sustained by the nerve supply of the clavicular portion of the pectoralis major which has been found occasionally to be supplied by a twig from the circumflex, thus having the same nerve distribution as the deltoid. Furthermore, normally it receives its innervation from that portion of the external anterior thoracic nerve which receives its fibers from the fifth segment.

Consequently, it is not surprising, but rather to be expected, that the clavicular head of the pectoralis major should resume its former association and along with the clavicular head of the deltoid serve in part, at least, a like function, namely, as an elevator of the humerus from above 115 degrees.

CONCLUSIONS

1. The mechanism of elevation of the arm may be divided into three physiologic cycles in which the deltoid and supraspinatus are alternatingly motofacient and nonmotofacient. The first cycle of elevation is accomplished by the action of the deltoid and supraspinatus, raising the humerus to about 60 degrees, and accompanied by slight rotation of the scapula; the second cycle is accomplished principally by rotation of the scapula due to the action of the serratus magnus, trapezius, rhomboideii, and levator angulii scapulae, which raises the arm to approximately 115 degrees, and is accompanied by slight elevation of the humerus; and the third cycle is accomplished by elevation of the humerus from 115 degrees to a straight angle due to the action of the deltoid and supraspinatus in which there is only slight rotation of the scapula.

2. The older view that the deltoid raises the arm to approximately a right angle, after which its action ceases and the movement of elevation is completed by rotation of the scapula, is not sustained. The deltoid elevates the arm both in the initial and final stages of elevation.

3. Associated with the deltoid and supraspinatus in the final stage of elevation are the coracobrachialis and the clavicular head of the pectoralis major. The latter, phylogenetically, is closely related to the deltoid and in its development may be considered as a migration of the deltoid fibers. Hence, it is not surprising that this muscle should resume its former association in function and assist in elevation of the humerus from above 115 degrees.

471 Park Avenue.

News and Comment

ANNUAL MEETING OF THE AMERICAN MEDICO-PSYCHOLOGICAL ASSOCIATION

At the seventy-sixth annual meeting of the American Medico-Psychological Association the following officers were elected: president, Dr. Owen Copp, Philadelphia; vice president, Dr. Sanger Brown, Kenilworth, Ill.; secretary-treasurer, Dr. H. W. Mitchell, Warren, Pa. Dr. George M. Kline of the Massachusetts State Commission of Mental Diseases is chairman of the entertainment committee, and Dr. Stanley Abbott is chairman of the program committee.

The next annual meeting is to be held at Hotel Somerset, Commonwealth Avenue, Boston, May 31, June 1, 2 and 3, 1921.

In addition to the annual meeting the association will, during the coming year, take up the question of revision of the constitution. Among other changes suggested is change of name of the association from the American Medico-Psychological Association to the American Psychiatric Association.

Abstracts from Current Literature

THE SPIROCHETE OF GENERAL PARALYSIS. Levaditi and Maria, Ann. de l'Inst. Pasteur **33**:741, 1919.

This study on the spirochete of general paralysis represents the continuation of experimental work interrupted by the war.

In 1913 (Compt. rend. Acad. d. sc. **157**:864, 1913; ibid. **158**:1595, 1914) the authors, in collaboration with Danulesco, succeeded in transmitting spirochetes from a case of paresis to the rabbit, and in producing typical lesions. Several passages were made and comparative biologic and pathologic studies were made of this virus and the virus of human syphilis, cutaneous and visceral. The study showed such manifest differences between the two parasites that the conclusion was inevitable that the spirochete of general paralysis must be regarded as a distinct, neurotropic variety of *Spirochaeta pallida,* and that the pathologic agent of syphilis is present in the circulation of the paretic.

A complete review of the literature is made regarding the demonstration of the spirochete in the cerebral cortex, the blood and the spinal fluid in cases of paresis. The results of all these studies show that the spirochete is present in the cerebral cortex, blood, and spinal fluid in all cases of paresis, irrespective of the clinical manifestations, progress and severity of the disease; and that the presence of spirochetes can be demonstrated either by microscopic examination (brain) or by inoculation experiments. Although the positive results of these experiments are by no means constant, they show a remarkable increase with the increased number of experiments and the abandonment of postmortem studies for observation in vivo and the use of the ultramicroscope.

The demonstration of spirochetes in the cerebral cortex of paretics is almost a constant finding, but is less frequent in the blood and spinal fluid. In the blood and spinal fluid the presence of the parasite is probably intermittent and transitory.

Clinical observations are presented which suggest the existence of a neurotropic strain of *Spirochaeta pallida.* These observations are: 1. The fact that early syphilitic manifestations in paretics and tabetics are slight, transient or absent, and the tertiary manifestations in the skin and viscera exceedingly rare. Fournier's observations on 243 cases of severe syphilis are quoted. These cases were observed by Fournier for a number of years and not one developed paresis or tabes. 2. The rarity of paresis among natives of tropical countries where syphilis occurs in a severe form and is treated insufficiently or not at all. 3. The existence of conjugal or familial neurosyphilis; although the authors state that the number of such instances are not numerous considering the frequency of tabes and paresis. A number of the reported cases of conjugal neurosyphilis are presented.

Two strains of spirochetes were studied. One was obtained by Touffi from a patient with cutaneous syphilis, the other by the authors from the blood of a paretic. The former is termed dermotropic virus and the latter neurotropic virus.

The viruses show a difference in their incubation period, in the character of the lesions, the progress and virulence of the disease. The incubation period of the neurotropic virus is much longer (55, 60, 77, 94 days) than the dermotropic virus (43, 46, 49, 60, 77 days).

Character of Lesions: The dermatropic virus produces a papulosquamous erosion. Microscopic examination reveals erosion and desquamatous superficial ulceration of the epidermis, vascular lesions without periarteritis or endarteritis of the dermis, and slight infiltration. There is no conjunctival neoformation. The spirochete is found in the epithelial layer.

The neurotropic virus produces a papulosquamous erosion. Microscopic examination reveals erosion, desquamation and superficial ulceration of the epidermis, vascular lesions without periarteritis or endarteritis of the dermis, and slight infiltration. There is no conjunctival neoformation. The spirochete is found in the epithelial layer.

The dermotropic virus produces an indurated chancre. Microscopic examination reveals deep ulceration of the epidermis, peri-arteritis and endarteritis of the dermis, and intense infiltration. There is intense conjunctival neoformation. The spirochete is found in the deep layers.

Virulence: The two viruses show a striking difference in virulence. The dermotropic virus retains its virulence for the lower apes and the anthropoid apes even after many passages through the rabbit, tested during the years 1908-1914. Through the accidental inoculation of one of the assistants in the experiments, the authors were able to demonstrate that it continues to be pathogenic for the human subject as well, the local lesions, however, in the latter were mild, no secondary symptoms developed and the Wassermann reaction became positive long after the local lesion appeared.

The virulence of the neurotropic virus was studied in two cases of paresis, and it was found that this virus, which was pathogenic for the rabbit and apes and the human subject, was avirulent for the paretics. On the other hand, the neurotropic virus is avirulent for the ape, if inoculated after a single passage through the rabbit; similar results were obtained from experiments on the human subject.

Crossed inoculation experiments showed that rabbits which had recovered from infection with dermatropic virus and were vaccinated again this virus developed, when inoculated with neurotropic virus, the characteristic experimental papulosquamous lesion. The inverse experiment was positive.

The conclusion, therefore, appears justified that there is a biologic difference between the two parasites. Experiments conducted to justify this conclusion showed that rabbits after presenting the local lesion provoked by dermatropic or neurotropic virus, acquire a refractory condition with regard to a homologous spirochete, and retain their receptivity toward a heterogeneous virus. The dissemblance between the two viruses is at least as marked as that existing between the spirilla of Dutton and that of Obermayer, the pathogenic agents of African and European relapsing fever.

The question whether or not syphilitics destined to develop tabes or general paralysis are contaminated at the onset of their syphilis by a special spirochete, or whether this special type, owing to its residence in the central nervous system, is created as the result of a progressive adaptability, is one that is not easily determined. While some of the observations cited seem to indicate an original contamination, the authors believe that the two hypotheses are not only perfectly compatible, but rather tend to confirm the theory of a neurotropic virus. Among the numerous syphilitic germs, morphologically similar, but differing in virulence, which transmit syphilis in civilized countries as the result of old infection, there are some which are distingushed by a special aptitude to localize in the central nervous system. This does not imply that they are incapable of localizing in the skin or in certain viscera or in the cerebrospinal fluid. Otherwise we would not be able to understand their abiltiy to be transmitted from one individual to another by the cutaneous, mucous or circulatory route (hereditary). But they do seem from the very start to have a weak reaction on the skin and the mucous surfaces, as is evidenced by the transitory symptoms and their mildness as seen in the future paralytics or tabetics.

This neurotropic spirochete, however, is very early transformed into one whose affinity for the brain and spinal cord becomes more and more marked and almost exclusive, in that the parasites thrive in contact with the cerebrospinal substance. And it is precisely this type of spirochete which we have been studying and which we have compared with the dermatropic virus.

It also seems that the difference between the two viruses is not so great at the time of the initial contamination as it is when paresis or tabes is fully developed. In other words, the spirochete treponema that later becomes neurotropic protects the human subject against cutaneous and mucous syphilis, while the parasite already neurotropic does not immunize the rabbit against the dermatropic virus. In short, the budding of so-called parasyphilis appears to be due to a special kind of contamination by a type of spirochete with a more or less neurotropic affinity. This neurotropic character may be so definite that the lesions in the cerebral cortex and the spinal cord have nothing in common with those generally considered by pathologists as characteristic of syphilis. In fact, many who have studied the histology of paresis emphasize the lack of resemblance between the existing syphilitic change proper (endarteritis, gumma, meningitis, etc.) and those of paresis.

The question has assumed greater importance in recent years. The war and certain occupational diseases seem to show that nervous syphilis, apparently latent, may become active and progress rapidly into a galloping general paralysis. Our theory would seem to indicate that the lowered resistance of the cerebrospinal centers favor local pullulation of a latent neurotropic virus.

The authors realize the difficulties and the defects of the study. Many questions remain unsolved, prominent among them the inefficacy of various antisyphilitic treatments in general paralysis and tabes. This will be considered in a future publication. In the meanwhile they repeat their belief, expressed in 1914, that this inefficacy is not due to the resistance to arsenic of the neurotropic virus, since rabbits infected with that virus recovered just as well as animals infected with dermatropic virus (of Truffi), after intravenous injections of arsenobenzol.

Klauder, Philadelphia.

ADVERTENCIA DE LA REDACCION (EDITORIAL NOTE). Arch. de neurobiol., psic., fisiol., histol., neurol. y psiquiat. **1**:1, 1920.

The introductory word by the editor is of interest as it throws some light on the conditions under which scientific work is done in Spain at present.

The editor calls attention to the recent progress in neurology and psychiatry in Spain which is due to the scientific activity of a studious set of young men formed largely by association with Cajal and Simarro, and by the work and study in the clinics and laboratories of those nations most advanced in science. The editor notes that this progressive movement has taken place outside of the university which "does not consider these activities worthy of a chair."

"This journal, therefore, is founded to consolidate and organize this scientific movement to receive the work of Spanish masters and of the new generations of investigators and to make the work of Spanish students known in progressive lands."

ATAQUES EPILEPTICOS EN LA ESQUIZOFRENIA (EPILEPTIC FITS IN SCHIZOPHRENIA). José M. Sacristán, Ibid., p. 17.

The author offers nothing new or original in regard to epileptic convulsions in schizophrenia, but he reviews the latest articles on the subject giving a good bibliography and presents his own two cases as confirmatory of the Kraepelin-Bleuler theories: the metabolistic-toxic origin of the convulsions in schizophrenia.

LAS NEURITIS POSTGRIPALES (POSTINFLUENZAL NEURITIS). Jose M. de Villaverde, Ibid., p. 23.

This article contains a careful and detailed consideration of postinfluenzal neuritis and with right emphasizes the necessity of distinguishing between paralyses occurring from neuritis and those caused by nuclear foci. The large number of third and sixth nerve involvement, without any lethargic symptoms, is interesting.

UN CASO DE TUMOR DEL CUERPO CALLOSO SIN DISPRAXIA IZQUIERDA (A CASE OF TUMOR OF THE CORPUS CALLOSUM WITHOUT LEFT DYSPRAXIA). Gounzalo R. Lafora, Ibid., p. 45.

The comparative rarity of tumors of the corpus callosum renders every well studied case of interest especially if, as in the case presented, some symptom or symptoms are unexpected. The author has made a careful analysis of his case and it is presented with an outline of the work done by Liepmann, Déjerine and others.

ALGUNOS OBSERVACIONES SOBRE LA HISTOGENESIS DE LA NEUROGLIA EN EL BULBO OLFATIVO (SOME OBSERVATIONS ON THE HISTOGENESIS OF THE NEUROGLIA IN THE OLFACTORY BULB). F. de Castro, Ibid., p. 49.

The author treats briefly the dual theory of the origin of the neuroglia: that it is produced in part by the ectodermic tissues and in part by the mesodermic, and the unitarian or ectodermal theory of the origin of glial tissues, and from his own research ranges himself on the side of the ectodermal theory.

His work was done on the olfactory bulb of the dog and cat at birth, and at 10, 20 and 40 days. He made use of Cajal's gold sublimate method and the same master's uranium-formol method. His conclusions are:

1. In the olfactory bulb of the new-born dog and cat all the phases of the evolution of the primitive epithelial cell can be followed up to the production of the glial cell, thus confirming Cajal's theory that the neuroglial cells are the product of epithelial cells or spongioblasts adapted to the environment.
2. In the molecular zone twin glial cells or groups are numerous, showing the activity of the proliferation of the glia at this period of its development.
3. The neuroglia of animals of a few days' existence is polarized in all the different layers and maintains its orientation toward the bulbar surface.
4. When the genuine epithelial cell is in relation to the ventricular cavity it shows a vigorous internal expansion, while the external prolongation is fine, free in its course and terminates in a point without ever being implanted on the pia mater.
5. In all the cells of the ependymal wall the centrosome, Golgi's reticulum and the mitochondrial granules are lodged in the interior of the cell or looking toward the ventricular cavity.

6. As soon as the epithelial element begins to detach itself and leave the ventricle, the crest emigrates to the surface, its exterior becomes netlike or branched and insertion into the vessels takes place at the free extremities. In the meantime the internal expansion becomes slender and finally atrophic and disappears.

7. In cells recently detached the centrosome and apparatus of Golgi have changed their position in the cell, both being in the exterior segment of the cell or in the direction of the largest peripheral branch.

8. The attraction of the vessels for the neuroglial protoplasma in the formation of glial feet (suckers, chupadores) must be relatively slight or nil. Probably previous trophic exigencies of the neuroglial cell exist in modeling the neuroglial feet.

9. In the evolution of the epithelial cell the phenomenon of retrograde atrophy of the radial stalk and also some of its collaterals is specially noted.

10. The collaterals which arise in the course of the radial stalk take their origin in the most robust side branches (vellosidades) on the stalk referred to and singularly enough in those placed peripherally. The formation of the collaterals is a manifestation of the most advanced period in the evolution of the neuroglia, appearing, probably, preceding the retrograde atrophy of the peripheral expansion, and being a necessary formation in the extreme differentiation of the glial element.

11. Some of the arguments exposed in the preceding conclusions in regard to the epithelial cell of the mammifers may be noted also in the same body in the philogenetic series. The batrachians and reptiles form an exception. In these vertebrates the vascular insertions take place commonly on peripheral capillaries and are established in collaterals coming from the stalk.

NUEVOS METODOS DE ANALISIS DEL LIQUIDO CEFALORAQUIDEO (NEW METHODS OF ANALYSIS OF THE CEREBROSPINAL FLUID). Gounzalo R. Lafora and Miguel Prados y Such, Ibid., p. 80.

This article on new methods of analysis of the cerebrospinal fluid does not give any methods peculiar to the authors but considers the technic and the value of the mastic reaction as done by Emmanuel and modified by Kafka and the Prussian blue test published by Kirchberg in 1917.

In the majority of their cases the authors found that the mastic reaction coincided with the Lange gold test. The gold reaction from blue to colorless (3 to 5) always gives positive reactions in the mastic, almost always when the curve is low (color 5 colorless) the mastic reaction is positive in four tubes (+ + + +). In median Lange curves (3443321100 or 4433321000) the mastic is positive only in two or three tubes. The minimum limit of Lange's reaction in which a mastic reaction is found corresponds to the curves in which some tube is blue (222321000) or color 3. One exception only was observed to this rule in the case of a probable brain abscess caused by a suppurating mastoid.

The mastic reaction does not answer as a diagnostic means for differentiating general paralysis of the insane from tabes and cerebral syphilis, at least, if used by the Emmanuel method. In optic neuritis the mastic coincides with Lange's in being negative. In nonsyphilitic diseases (epilepsy, dementia praecox, angioneurotic edema) Lange and mastic coincide in negativity. In the case of supposed brain abscess mastic was positive (+ +) and Lange almost negative (2211000000).

The authors have found Prussian blue uncertain in its results and altogether inferior to the mastic.

GURD, Ann Arbor, Mich.

ZUR ENTWICKELUNG UND PATHOLOGISCHEN ANATOMIE DER RAUTENPLEXUS (THE EMBRYOLOGY AND THE PATHOLOGIC ANATOMY OF THE CHOROID PLEXUS OF THE FOURTH VENTRICLE). C. VON MONAKOW, Schweizer Arch. f. Neurol. u. Psych. **5**:378, 1919.

The contribution represents the results of a further study of the relation that the choroid plexuses bear to diseases of the central nervous system. In a previous article (*Arch. Neurol. & Psychiat.* **3**:182, 1920) von Monakow and Kitabayashi reported extensive degenerative changes in the choroid plexuses of the hemispheres in severe forms of psychic disturbances, particularly those exhibiting schizophrenic manifestations.

Complete serial sections of the brains of about fifteen human embryos from 1 to 20 cm. in length, representing from the second to the fourth fetal months, and sections from more fully developed fetuses and from the brains of children were prepared. It was noted that the ventricles of the oblongata could be divided into completely separated compartments, one medial and two lateral. These spaces contain the medial and lateral plexuses of this ventricle; they are lined with ependymal cells, are completely separated from each other, and communicate nowhere with the subarachnoid spaces. In a fetus 1 cm. in length there are accordingly no morphologically preformed foramina of Magendie or of Luschka. In one of 2.4 cm. we have as yet no evidence of a choroid plexus. There is noted an accumulation of hematopoietic cells representing the anlage of the plexus; the primitive folds of the lateral plates at this time assume a villous character. The choroid plexus of the fourth ventricle is directed medially from the region of the lateral recess, while that of the lateral diverticulum arises from the region of the corpus restiforme and is directed ventrolaterad. The villi, at this stage, are covered with well differentiated, glandlike cells derived from the ependymal matrix, but are as yet almost entirely free from blood vessels. Even thus early, protoplasmic bodies are liberated from these cells and enter the ventricular spaces. Monakow is of the opinion that these deliver material, through the medium of ventricular fluid, which is necessary for the further tectonic development of the oblongata. In embryos of 3 cm. length the bilateral anlage of the cerebellum is readily discerned; in those of 8 cm. the flocculus shows its appearance. Nowhere do the chambers of the lateral diverticulum and that of the fourth ventricle proper appear to communicate.

In a child of 1 year the anatomic relations are exactly the same as in an adult, save that the blood vessels are smaller and the pericapillary connective tissue more delicate. Monakow insists that serial sections even of the adult brain show that the location of the foramina of Luschka represent merely the place of entry of the larger blood vessels into the plexuses; a preformed communication between the ventricles and the subarachnoid spaces does not exist. Also, in all probability, the lateral diverticulum and the fourth ventricle do not communicate directly. The wall of the diverticulum consists of (*a*) ependymal cells, mostly of a single layer, arranged in the form of pallisades, (*b*) subependymal tissue in which glial elements, small nerve cells, unmedullated nerve fibers, and molecular substances occur, and (*c*) a dense outer layer which separates these structures from the subarachnoid space.

Monakow sees a great biologic significance in the physiologic-pathologic relation of the tuberculum acusticum, the ventral acustic ganglion, and the lateral walls of the flocculus to the lateral diverticulum. The lateral and caudal portion of the acoustic tubercle, as well as of the ventral ganglion, are covered completely with ependymal cells, permitting direct contact with the villi of the choroid plexus, providing nutritive material in the way of ferments; on the other hand, the medial and anterior portion of the acustic region is located outside of the diverticulum, is free from ependyma but clothed with vascular arachnoid, and is consequently nourished with arterial blood, providing it with oxygen, salt, etc.

It is quite possible that the frequent evidences of irritation of the auditory apparatus in the way of tinnitus are concerned with abnormal composition or pressure of the fluid in the lateral diverticulum or pathologic changes of the plexus itself.

The author repeats some of the findings noted in his previous paper on disease of the choroid plexus as a whole, and then proceeds to discuss the significance of localized and primarily meningitic lesions of the plexus of the fourth ventricle. In cases of deafmutism, with or without idiocy, pathologic changes appear with greater frequency in the plexus in the lateral diverticulum, leading to atrophic conditions of the neighboring portion of the oblongata and the cerebellum. A third form of disturbance is the result of hypoplasia arising in the earliest embryonal development. A fourth variety represents diffuse and irregular atrophic changes occurring mostly in the plexus in conditions of general arteriosclerosis, senility, and renal sclerosis.

Monakow believes that it is not at all improbable that these disturbances may lead to mechanical irritation, resulting in abnormal sensations and leading ultimately to illusions, hallucinations, and schizophrenic phenomena. An analagous condition has been reported on numerous occasions in the sensation of formication, tickling, and the like, occurring in the opposite extremity when the central convolutions are touched in unanesthetized patients. Doubtless, also, noises may result from irritation of the primary acoustic centers, and abnormal sensations of taste from irritation of the glossopharyngeal area. It seems not amiss to venture the statement in the form of an hypothesis that biochemical reactions through the medium of abnormally constituted fluids may evoke responses, similar to those produced by the grosser mechanical irritations of the cortex.

Woltman, Rochester, Minn.

SUR LE PHENOMENE DE STRUEMPELL (STRUEMPELL'S PHENOMENON). Noica, Rev. neurol. **35**:567 (July) 1919.

Strümpell's phenomenon is briefly described. When the leg of a patient with spasticity is flexed on the thigh there is a flexion of the foot on the ankle, and frequently there is an extension of the great toe not unlike the Babinski phenomenon. The entire sign is more pronounced if the examiner exerts pressure on the knee of the subject without, however, preventing the movement at the knee. Strümpell himself never explained the mechanism of the movement. Babinski and Jarkowski in 1914 regarded the phenomenon as a conjugate movement due to mechanical traction of the aponeurosis in spastic conditions. Marie and Foix, on the contrary, 1916, regarded the sign as due to "active and associated movements" of the anterior leg muscles such as the vastus externus, the inner part of the soleus, etc.; in brief, the movements of synergic muscles; and as such they regard all so-called conjugate movements, styling them, "syncinetic movements of coordination." These they think are the result of the automatic functioning of the spinal cord freed from the inhibitions of the higher centers, i. e., they are the function of medullary automatism.

This difference of opinion between Babinski and Jarkowski on the one hand, and Marie and Foix on the other, is what stimulated the author to a further study of the Strümpell phenomenon. He made close observation by exerting movements of flexion and extension of the leg and the thigh in the cadaver (after "working out" by frequent movements the rigidity of the joints involved) in a normal boy of 15 and in a normal adult.

In his experiments on the cadaver he confirms Babinski and Jarkowski's opinion that the flexion or extension of one segment (the leg) is accompanied by flexion and extension of the other segment (the foot) and that this is purely mechanical. But he establishes that this phenomenon is not due to any part played by the aponeurosis or tendons of the leg, but aside from the purely mechanical condition, it is accompanied by, or due to, active muscular contraction according to the view of Marie and Foix.

In his experiments with a boy of 15 and with an adult, he comes to the conclusion that we find in normal subjects, especially in young persons, a phenomenon which closely resembles, if it is not identical to, the so-called phenomenon of Strümpell. If, then, a phenomenon analogous to that of Strümpell may be seen in normal persons, and especially in the young, is it equivalent to conjugate movements such as Babinski and Jarkowski conceive them, or is it equivalent to automatic movements of the cord such as Marie and Foix conceive?

The author then analyzes the phenomenon of Strümpell in abnormal or sick persons. He cites the case of a syphilitic patient who had vertigo, double optic neuritis, spasticity in the lower extremities, exaggerated tendon reflexes, sensory disturbances in the feet, lost cutaneous reflexes and a typical Strümpell sign. Yet this patient did not exhibit the great toe sign of Babinski, or the defensive movement of Marie and Foix. That is, there was no automatic cord movement, and yet the Strümpell phenomenon was present. From this it may be concluded that, while usually in spastic conditions, the Babinski, Marie and Foix, and Strümpell signs are all present, yet the latter is not constantly present. The absence of the coexistence of the phenomenon of Strümpell and movements of defense in paraplegics would lead us to think the two phenomena may be different.

The author then emphasizes the condition under which the Strümpell sign is observed; he says the Strümpell sign only obtains in hemiplegics or paraplegics when there exists sufficient muscular power to flex the knee and the foot.

On the basis of observations made on hemiplegics, paraplegics and patients suffering a lesion of the sciatic nerve, the writer regards the phenomenon of Strümpell as distinct from the phenomenon of defense of the physiologists, or the medullary automatism of Marie and Foix.

He adds that he has failed to obtain the Strümpell sign in certain hemiplegics, not because of any pathology in the pyramidal tract, but because of atrophy of the leg muscles in which the atrophy was marked by abundant adipose tissue.

In his final conclusion, the author regards the Strümpell sign as a normal associated movement, modified by the loss of motility of the extension of the leg, and by a partial preservation of the movements of the anterior muscles of the leg. He also believes that synergic voluntary movements are more easily executed than isolated voluntary movements and that isolated voluntary movements occur later in the development of the individual than synergic voluntary movements. Voluntary isolated movements are brought about by act of will, and then by training; but the will is always necessary, not only to execute the movement, but at the same time to inhibit an associated movement. Therefore, a lesion of the pyramidal tract, which partially inhibits voluntary motility, diminishes at the same time, isolated movements. We revert as it were, to a state of infancy; to a period when we have not yet undertaken to dissociate the complexity of spontaneous movements. We are in a state of being able to execute nothing but synergic movements. Such are the conditions under which the phenomenon of Strümpell exhibits itself.

JONES, Detroit.

SUGGESTIONS AND SUGGESTIBILITY. E. PRIDEAUX, Brain **42**:291 (Jan.) 1920.

Prideaux discusses suggestion and suggestibility. Suggestion is defined as the conveyance and acceptance of an idea without logical grounds on the part of the person who accepts the suggestion. It is largely an affective process, and conviction in the mind of a patient is produced when the suggestion advanced is in harmony with instinctive tendencies, sentiments, interests and complexes and a strong affective force is aroused. This holds for normal suggestion, but is even more pronounced when it is abnormal, as in hysteria. Here the affective currents are stronger and the contrary ones weaker.

Four forms of suggestibility are recognized: First, individual, which varies in different persons, without regard either to the nature of the suggestion or of the suggestor and is explained by the "varying degree in which the egoistic instinctive tendencies are developed, and the manner in which the sentiments have become organized to form ideas and act as contrary forces." Second, conditional, which varies in the same person at different times and under different conditions. This depends "on the relation of the suggested idea to that state." Third, specific, which has reference to a particular system of ideas only. This in itself is proof of the affective nature of the process. It is a well-known fact that a person is particularly receptive to ideas which are pleasing to him and which satisfy his egoistic instinctive tendencies. Fourth, personal, which is effected by the reaction of the suggestee to the suggestor and is more or less dependent on authority, prestige, sympathy, respect, confidence and the like.

The mode of response to suggestion may be classified into positive, negative and neutral. In the positive type the suggestion is accepted not by inhibition of other ideas, but because it becomes endowed with a strong emotional force that overcomes all contrary forces. When the suggestion is opposed, a negative response is obtained. This occurs when the suggested idea is not only "incompatible with preformed sentiments and interests, but arouses contrary emotions and sentiments." The author sees in the negativism of praecox an exaggerated example of such a response. The response is neutral when the suggestion is refused. Here we may be dealing with a person who holds strong principles and ideals or one like an imbecile who has no preformed interests or complexes to which the idea may attach itself and, therefore, the suggestion cannot make sufficient impression, or the suggestion may call forth incompatible forces which neutralize each other.

The author makes some practical psychotherapeutic suggestions. The physician may, and of course as a matter of fact does, use his prestige and authority to stimulate the latent suggestibility of the patient. If the patient is only suggestible in a particular sense; then the suggestions must be arranged and presented so that incompatibilities will be avoided. Favorable effective states may be created. Thus surprise will "clear the mind" and make a response more likely. If fear exists it may be neutralized by inducing anger, for these two emotions "tend to exclude one another from simultaneous activity."

STRECKER, Philadelphia.

SUICIDE AMONG SOLDIERS RECEIVED AT THE HOSPITAL FOR PSYCHIATRIC CASES AT MOMBELLO. GIUSEPPE SACCHINI, Arch. di antrop. crim. **29**: Pt. 2.

A statistical study of the soldiers received at the psychiatric hospital reveals the fact that suicidal attempts are more frequent among mentally diseased soldiers than among civilians. War, by its violent reactions, renders less repugnant and even familiar the idea of death. For one type of person suicide becomes the preferred remedy for all his anguish.

Detailed histories of fifty cases are given. In only one case was the result fatal. Fourteen of the men had previously attempted suicide in civil life. The hereditary taint among the fifty patients was marked, alcoholism being present in 32 per cent., suicide in 30 per cent., psychopathy in 16 per cent., neuropathy in 16 per cent., etc. In only four patients was there no taint. Ten were syphilitic and three tuberculous, while in three others the patient feared one or other of the above diseases. Six patients were convalescing from a trauma or acute disease while in the past history of twenty others there was a history of serious illness followed by neurasthenic states, intermittent headache, irritability, weakening of the critical power, etc. The exciting cause of the suicidal attempt was "tired of living" in eight patients; hypochondriac preoccupations in nine; disappointment in love in four; and in six there was no assignable cause. Among the definitely pathologic causes were: attacks of unconsciousness in seven, brief and partial blurring of consciousness in five and grave mental disturbance in three. Among the strictly military reasons were fear of censure by superiors and fear to return to the front. During their convalescence eighteen were in a melancholic state and fifteen others showed definite mental pathology. The predominating forms of mental disturbance were neurasthenia, epilepsy, dementia praecox and feeblemindedness. As to the means of committing suicide, twenty-two chose poison, four firearms, five cutting instruments, nine threw themselves to the ground from a height, five threw themselves into the water, two under heavy vehicles and seven attempted hanging. Two made two or more attempts by one or more methods. Seven had previously been refused for military service, eight had criminal records and almost all showed degenerative stigmas. The recruiting of such defectives naturally markedly increases the percentage of all crimes among soldiers.

The necessity of foreseeing the consequences of congenital and acquired mental abnormality has been widely recognized in this war with a resultant increase in the military hospitals for the observation and cure of alienated soldiers.

The article is a careful analysis of the conditions leading up to attempts at suicide and an argument for the abnormal basis in the vast majority of cases. It is also an argument for the more careful selection of recruits, a lesson learned early in the late war by the United States military authorities.

Van Epps, Iowa City.

LE TARTRATE BORICO-POTASSIQUE ET LA MEDICATION BOREE DANS LE TRAITEMENT DE L'EPILEPSIE (BORIC POTASSIUM TARTRATE AND BORON MEDICATION IN EPILEPSY). Pierre Marie, Crouzon and Bouttier, Bull. de l'Acad. de méd. **83**:504 (June 1) 1920.

Thirty years ago Gowers extolled the use of borax (sodium borate) in epilepsy. Chemical impurities (?) of this drug have rendered results with it poor or uncertain. Therefore the authors have experimented with other boron salts using chiefly boric potassium tartrate, otherwise known as boric emetic and also as soluble cream of tartar; with the following atomic formula: $Co_2H\text{-}CHOH\text{-}CHO\ (B_0\ O)\text{-}CO_2K$, and have watched the effect of this drug in grám doses three times a day on patients at Salpêtrière, who were "simple epileptics"—i. e., nonalienated They have used sodium tetraborate in other series.

They chose ten female epileptics who showed attacks maximal in frequency and intensity. For the months that the drug was administered and for the month preceding these patients were careful observed, day and night, so that all attacks might be recorded. (They had all been without bromids for either years or months.) The apparently beneficial effects of the drug is shown by the fact that these ten patients during the month preceding the administration of the drug averaged thirty and one-half attacks per month each; while during the period of treatment, three months, they averaged eighteen attacks each during the first month, eleven and one-half during the second and nine during the third. The writers not only report benefit in that the attacks were reduced in number but a greater portion of the attacks were vertiginous in nature rather than convulsive.

The drug is well borne and the occasional gastric disturbance encountered (diarrhea and vomiting) respond to its temporary withdrawal. Especially conspicuous is the total absence of depressive action on the psyche. Acne has been induced in some cases but not in a degree comparable to that caused by the bromids. Equally satisfactory effects have been obtained in children between 7 and 15 years of age. The authors have obtained benefit even in traumatic epilepsy and in epilepsy caused by cerebral tumor.

The therapeutic effects of bromids have especially for their basis the diminution of the reactional power of the nerve centers. For boric potassium tartrate it seems to be otherwise. One has the impression that it is on the lesion which is causing the epilepsy that the effect is gained. One is tempted to ask whether it is not by a sort of catalytic action that a small dose of boron determines such notable effects.

The conclusions are: 1. Boron medication plays an anti-epileptic rôle quite equal to that of bromin medication. 2. Boric potassium tartrate is superior to the other boron salts for the purpose.

Davis, New York.

CRIMES DUE TO THE WAR NEUROSES. Prof. Leone Lattes, Arch. di antrop. crim. **29**: Pt. 1-2.

The war neuroses as efficient causes of criminal acts have not been given sufficient recognition in comparison with other psychopathic states. After excluding all the cases in which "shell-shock" has been used as an excuse there remains a definite number which may be termed the "crimes of the psychoneurotics." These merit a more general consideration than commonly accorded because delicate questions of justice, humanity and social prophylaxis are involved. The difficulty arises not during the acute symptoms, but later when the convalescence is incomplete.

A detailed history is given of fourteen cases. In one the crime was homicidal attempt, in four desertion and in nine insubordination. Of the above cases two gave a history of morbid heredity and two of previous morbid symptoms. The relations between the crime and the neurosis is one of dependence and not of concomitance. The personality has been changed so that crime becomes possible. In almost all of the patients the previous conduct had been good and occasionally even distinguished. Only one had a previous military sentence and none a civil record. The typical offense of the psychoneurotic is insubordination based on a morbid irascibility and lack of inhibition. As the result of some innocent remark or reproof the patient reacts excessively and loses self control. The clinical evaluation of these facts is simple; the medicolegal is more difficult. The difficulty arises in the fact that the crime is dependent on a morbid alteration, the result of the injury to the mind and not on a constitutional psychic anomaly. In the interpretation of the code the morbid state of the accused at the time of the act nullifies the accusation. The military code is essentially preventive in scope. But rising above the restrictive view of punitive practice is that of social preservation. No doubt the symptoms of the psychoneurotic may be confused with criminal tendencies for the responsibility is intact, but the opposite mistake is more frequent and more embarrassing. The remedy is mainly preventive, namely, the recognition of the war neuroses and the establishment of cure before return to duty. This, however, demands an individualization in treatment and a stability of judgment rare in the vortices of war.

Van Epps, Iowa City.

LE POLYMORPHISME DE L'ENCEPHALITE EPIDEMIQUE, CLASSIFICATION DES FORMES CLINIQUES (THE POLYMORPHISM OF EPIDEMIC ENCEPHALITIS WITH A CLASSIFICATION OF CLINICAL FORMS). Henri Roger, Progrès méd. **35**:247 (June 5) 1920.

The author first points out the polymorphism of epidemic encephalitis and this is sufficiently emphasized by the classification which he then proceeds to formulate.

Apart from distinctions based on the soil in which the disease evolves (infantile form, senile form, gravid form) or apart from distinctions resting on anatomy (peduncular, bulbar, spinal or even polyneuritic forms) or apart from distinctions based on the course (acute, subacute, chronic, ambulatory or relapsing forms), he proposes, on a clinical basis and following the methods of Grasset, to distinguish forms due to exaggeration, deficiency or vitiation of nervous system functions: in other words, types in terms of hyperfunction, hypofunction and parafunction. On this basis he makes the following classification:

1. Motor Forms:
   (*a*) Hyper: myoclonic, choreo-ataxic, convulsive, tetanic.
   (*b*) Hypo: paraplegic type (of Sicard), polyneuritic (variations and combinations under (*a*) and (*b*) introduce the syndrome Millard-Gubler form, the oculomyochonic form, the ophthalmoplegic form).
   (*c*) Para: Parkinsonian or myotonic.
2. Sensory forms "of which we know only the hyper type, the algic."
3. Psychic forms.
   (*a*) Hyper: delirious, mental.
   (*b*) Hypo: with psychic depression.
4. Forms resting on the functioning of the sleep center.
   (*a*) Hyper: somnolent or lethargic.
   (*b*) Hypo: insomnious.

Davis, New York

PSYCHOSIS ASSOCIATED WITH TETANY. Albert M. Barrett, Am. J. Insan. **76**:373 (April) 1920.

Barrett had the unusual opportunity of studying two cases of tetany both of which came to necropsy. Two men, one aged 58 and one 46, presented classic neurologic symptoms of tetany. Mentally there were varying degrees of clouded consciousness at times amounting to stupor, depression and apprehension, auditory and visual hallucinosis. Of great interest is the fact that these symptoms appeared at irregular intervals and were succeeded by comparatively clear periods. In one case the relationship between an increase in the mental disturbance and constipation with excessive indican is significant.

Postmortem studies showed the effect of intense toxicity on the brain and cord. In both cases the pituitary body and thyroid were cystic. In one instance one parathyroid was independent, and there was parathyroid tissue in the substance of the thyroid. In the other three parathyroid were found. In neither case were there pathologic changes in these glands.

Both of Barrett's cases had numerous cysts of the pituitary glands. Here is additional evidence of the connection between these bodies and the parathyroids. It has already been noted that the hypophysis enlarges after thyroidectomy and parathyroidectomy, and tetany symptoms consequent on parathyroid removal in cats have been made to disappear by pituitary injection.

The author concludes that there is no specific tetany psychosis but that both the neuromuscular and the psychotic syndromes result from a toxic process affecting the central nervous system.

Strecker, Philadelphia.

Society Transactions

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Meeting, May 20, 1920

E. W. TAYLOR, M.D., *President*

REFLEX PHENOMENA IN THE CONTRACTURE STAGE OF PERIPHERAL FACIAL PARALYSIS. Presented by DR. A. MYERSON.

Following the acute phase of peripheral facial paralysis, of whatever origin, there is in the cases that partly recover a phase of contracture. In this period, which comes on gradually after six months or more, the patient superficially presents a picture quite the reverse of the acute phase in that the nasolabial fold on the affected side is more prominent and the aperture of the eye is narrower. There is usually some weakness of the face, with so-called associated movements, so that when the affected eye is closed the corner of the mouth moves with it.

The new phenomena described (illustrated on a patient) are: (1) when an electrode (either G. or F.) is placed on the affected brow and the current turned on, the corner of the mouth twitches before the brow contracts, although the direct excitability of the nerve is lessened; (2) tapping the forehead or the bridge of the nose causes a contracture (reflex) of the affected corner of the mouth.

These phenomena are absent on the sound side and in all other conditions. They seem to be specific to the contracture phase of the peripheral facial paralysis and relate to changes in the nucleus whereby excitability is increased and made diffuse.

THE DIFFERENTIAL DIAGNOSIS OF HYPERTHYROIDISM. Presented by DR. J. H. MEANS.

Overactivity of the thyroid gland is of interest to the neurologist, not only because of the numerous clinical nervous manifestations to which it gives rise, but also from the point of view of functional pathology.

The work of Cannon and his collaborators has clearly shown an intimate relationship between the thyroid gland and the sympathetic nervous system. also between the suprarenal and the thyroid. Secretory activity of the latter can be shown to occur if the sympathetic nerve is stimulated or if epinephrin is injected, and augmentation of the pressor action of epinephrin takes place on a sympathetic stimulation. Furthermore, a condition in many respects like exophthalmic goiter has been produced in cats by the anastomosis of the phrenic with the cervical sympathetic nerve.

The work of L. B. Wilson is also interesting in this connection. This investigator found in the cervical sympathetic ganglions, which had been removed from patients with exophthalmic goiter, definite lesions of the ganglion cells which he regarded as causally related to the morbid process in the thyroid.

The fully developed case of exophthalmic goiter presents no difficulties in the matter of diagnosis. The neurologist, however, is frequently confronted with patients presenting atypical or suggestive symptoms of hyperthyroidism which he may experience difficulty in interpreting. The differential diagnosis of the milder grades of hyperthyroidism may be no easy matter. As a rule, the differential diagnosis will be from various psychoneuroses, cardiac neuroses, effort syndrome, etc., and from early tuberculosis.

Of late years certain rather technical laboratory tests have been helpful in differentiating true hyperthyroidism from other types of disorder. Probably the most useful of these is the determination of the so-called basal metabolism. One of the chief functions of the thyroid gland is to preside over the rate of heat production of the animal body. An increase in heat production occurs in hyperthyroidism and a decrease in hypothyroidism. The determination of the combustion level, therefore, serves as a functional test of the thyroid. As such it is useful not only in estimating the relative degree of hyperthyroidism in a case of exophthalmic goiter, but it also serves as a means of differential diagnosis in borderline cases, the metabolism being elevated in true hyperthyroidism and not in the other disturbances with which hyperthyroidism may be confused.

The level of the heat production or basal metabolism can now be easily determined in the clinic by means of a respiration apparatus by the method of so-called indirect calorimetry.

The reaction to epinephrin has also been used to detect the presence of hyperthyroidism. It is undoubtedly positive in all cases of true hyperthyroidism, but, as Peabody has recently shown, it is also positive in such conditions as effort syndrome. It is therefore less reliable than the determination of the metabolism level as a diagnostic procedure.

The blood sugar curve has also been suggested. Aub and Davis, however, have shown that while an alimentary hyperglycemia is readily produced in hyperthyroid patients, the height of the curve bears little relationship to the intensity of the thyrotoxicosis.

In general we must regard these special tests as helpful, but we must, as in everything else, place the information gained by our five senses first. Nothing can supplant the importance of the clinical impression, but the special tests, as Dr. F. C. Shattuck has said, if we make them our slaves and not our masters, will be of distinct aid.

(The clinical records and charts of three patients which illustrated the value of metabolism studies both in differential diagnosis and in following the progress of exophthalmic goiter were then presented.)

CARDIAC NEUROSES. Presented by Dr. Paul D. White.

Cardiac neuroses are functional disorders of the nervous system attended primarily by cardiac symptoms. This subject is as old as the study of medicine itself; Hippocrates, Galen, and nearly all the other ancient masters wrote of it. It becomes obscured now and again and constantly needs reemphasis.

The recent war revealed the fact that the irritable heart of soldiers is of frequent occurrence. It is essentially a neurosis of the fatigue or anxiety type and should not be considered as heart disease. The frequency of its occurrence in the army was such that Dr. White's attention had since the war been called to its frequency in civilian life, particularly among young women. It seemed that nearly one-third of all the so-called "cardiac" patients under 40, whom he had been seeing, belong in this large group of cardiac neurotics. Obviously, therefore, the subject is very important.

The differential diagnosis of cardiac neurosis includes consideration of true heart disease, hyperthyroidism, low grade infections like tuberculosis and convalescence.

A working classification built on that of the psychoneuroses presented by Dr. Stanley Cobb is as follows:

1. Fatigue neurosis—so-called "neurasthenia."
2. Introspection neurosis—so-called "hypochondria."
3. Anxiety neurosis.
4. Substitution neurosis—so-called "hysteria."
5. Obsession neurosis—so-called "psychasthenia."

Ninety-five per cent. of all cases.

DISCUSSION

Dr. Morton Prince asked whether Dr. White had found any enlargement of the heart accompanying his cases of cardiac neurosis.

Dr. Paul D. White replied that patients who had cardiac neurosis pure and simple had no enlargement nor murmur. There was no sign of actual heart disease in any case of true uncomplicated cardiac neurosis.

Dr. Morton Prince said that he had examined the hearts of many thousands of men in the prime of life and found not only increased heart action (sometimes irregular), but also an enlargement of the heart. In a good many cases mitral systolic murmurs were present. Not being certain of the organic origin of these symptoms, Dr. Prince made a long series of investigations on several hundred men with the result, as was demonstrated, that he found the cardiac symptoms were due to the emotional excitement attending the examination. It became plainly evident that the enlargement of the heart, when present, was only the normal physiologic dilatation that occurs under excessive stimulation to enable the heart to do the work that it is called on to perform. In some cases the dilatation was so extreme as to permit some mitral valve leakage, thus causing the murmurs.

As a control experiment, a series of examinations was conducted under conditions in which the emotional excitement was eliminated, with the result that the previously observed cardiac symptoms disappeared. In another series of observations the anxiety was artificially stimulated as much as possible: in this series the symptoms were even more frequent. These observations were published in the *Medical Record* and the *Transactions of the Association of American Physicians,* and were among the first reports to show that the heart expands and contracts according to the work to be done and that this occurs under nervous stimulation.

The disturbances observed were of a temporary nature, but they are comparable to those observed in an anxiety neurosis of a chronic character. They might be called "acute anxiety neurosis." They are in principle what used to be called "irritable heart" when the emotional factor was overlooked. They should be considered not as the manifestations of an abnormal heart, but as the normal physiologic cardiac expression of abnormal anxiety. The anxiety is pathologic, not the cardiac manifestations.

Dr. A. Myerson asked Dr. Means whether in nontoxic goiters the metabolism varied. Dr. White brought up the point that cardiac neuroses were found in young people. Why are they not found in older people? Do such patients die, and if they do not spontaneously recover, how do they get well? Reassurance in his experience has little value. Does the condition disappear with early adult life and what is the mechanism of recovery?

Dr. Walter B. Swift said that a word ought to be said about the speech in cases of goiter. In 1918 a goiter zone was discovered in Cleveland and the speech studied in thirty cases. Each one of these patients was put through a series of some forty speech tests, making in all over 1,200 speech tests with tabulation of data on each. He was not ready to give full details of this paper, but investigation showed that goiter speech as a whole is diminished, slightly slow, of remarkably low pitch and harsh. In lay terms it might be called mannish when it occurs in women, whose natural speech is an octave higher than speech in men. In contradistinction, and as an item of differential diagnosis, the speech in neurasthenia is inclined to be fuller than usual in amount, increased in speed, high in pitch and rather quick and reflexlike in nature. The cause could not be found at first.

Dr. Morton Prince said that he had a strong suspicion that in many of Dr. White's cases anxiety was present. This is easily overlooked, but can be brought out readily by searching examination.

Dr. James B. Ayer said that he understood that when Dr. Goetsch returned from Saranac he said that about half the patients treated there for incipient tuberculosis were, in his opinion, cases of hyperthyroidism. It would seem to him reasonable that the asthenia of the tuberculous patient, coupled with temperature, might make a particularly susceptible sympathetic nervous system which would respond to the suprarenal test, giving the picture of hyperthyroidism. Dr. Ayer asked Dr. Means whether he still considered that there was a metabolic change which is characteristic of hyperpituitarism and hypopituitarism as well as hyperthyroidism and hypothyroidism.

Dr. W. E. Paul said in regard to thyroid changes that the hyperthyroid cases may become hypothyroid as time goes on. He had seen one or two and would be glad to hear of any others.

Dr. Henry Viets said that some one had suggested that in cases of encephalitis lethargica sleepiness may be due to some change in the pituitary gland. He wondered whether Dr. Means knew of any relation of metabolism to the marked lethargy of lethargic encephalitis.

Dr. Donald Gregg asked how it was possible to tell whether there was not some suprarenal disturbance in some cases in which a positive reaction with epinephrin was obtained. The thyroid disturbance is barred out, but how about the suprarenal cases? Do they also react very strongly?

Dr. J. H. Means, answering Dr. Myerson's question about nontoxic goiter, said metabolism is always normal in this condition. He thought there were two fairly definite diseases—exophthalmic goiter and adenoma. Exophthalmic is a uniform hyperplasia of the thyroid gland with hyperthyroidism. Any adenoma may apparently become toxic; then we may have a hyperthyroidism. Under those circumstances there is an elevation in the metabolism, but it is always accompanied by clinical manifestations. Goiter patients who are perfectly well except for the lump in the neck have no elevation.

Answering Dr. Ruggles' question about myxedema, Dr. Means said in general the clinical picture runs about parallel with the fall in metabolism. The fall is usually about from 25 per cent. to 33 per cent. The metabolism level is an indication of the amount of thyroid to give. He thought the dose required in individual cases varied a good deal. For example, he cited the case of a nurse who had an acute thyroiditis, an infectious process. She had had a non-toxic goiter for a number of years. It suddenly increased in size and became very tender, and she had a high temperature. The gland was removed surgically and on examination showed a definite inflammatory process—not exophthalmic goiter. About three weeks later Dr. Means found the metabolism was 15 per cent. The patient looked normal. He saw her again about four weeks later. In the meantime, she had developed typical myxedema. The drop in metabolism had preceded the development of clinical symptoms. After the administration of thyroid she became normal.

Answering Dr. Ayer's question, Dr. Means said that he had no personal experience with the Goetsch test. He believed that some of the cases that reacted to his test were not cases of hyperthyroidism, but were some kind of neuroses. He believed the thyroid glands were not abnormal, but that the vegetative nervous systems were unduly active, which is fairly distinct from true hyperthyroidism. Regarding pituitary conditions, there is a change in metabolism in pituitary disease. It is not so constant or so striking as in hyperthyroidism, but in active cases of overfunctioning of the pituitary gland there is sometimes an increase of about 40 per cent. and in some cases of hypopituitarism a slight reduction. There is no definite alteration in old acromegalics. When the process has died down the metabolism is generally normal.

Dr. Means answered Dr. Paul's question in the affirmative because recently he had studied a case of mild exophthalmic goiter. The patient's rate was + 50 per cent. only six weeks ago. She had all the symptoms of exophthalmic goiter. After three roentgen-ray treatments she came back to the hospital and he didn't recognize her. She presented a picture not characteristic of myxedema, but suggestive of it, and her metabolism was 12 per cent. below normal. She was given small doses of thyroid and became more nearly normal in appearance. This is the only patient he had ever seen who became definitely myxedemic after roentgen-ray treatments. Some of the postoperative cases that he had seen suggestive of myxedema had a slightly depressed metabolism.

Answering Dr. Viets' question, Dr. Means said he did not know of any observations that had been made on lethargic encephalitis.

Dr. Means could not answer Dr. Gregg's question. We know very little about primary disease of the suprarenals. He had one case of Addison's disease which showed no change. Cases of recognized suprarenal deficiency are very rare.

Dr. Paul D. White agreed with Dr. Prince except in regard to the effect of excitement on the heart size. The term "soldier's heart" is satisfactory. Anxiety undoubtedly plays a more prominent part than we realize. He recently saw a patient in whom the anxiety neurosis was undoubtedly a factor—a young woman with a disfiguring lupus vulgaris. She had typical cardiac neurosis symptoms. The effect of exercise and excitement on the heart is, of course, to increase the rate and force of the heart beat and, if the case is extreme, one also finds the presence of a systolic murmur. Immediately after exercise the heart is actually smaller than before, as proved by the roentgen-ray examination. The force of the heart is such that, on physical examination, the apex impulse may give an impression of dilatation. The murmur is commonly found at times of excitement, probably not to be explained however by dilatation of the mitral ring. His experience was too limited to tell what happens to these patients with cardiac neuroses as they grow old. They probably recover. An important point may be the relationship of cardiac neurosis to a latent production of arteriosclerosis. It is possible that one of the factors of arteriosclerosis is the constant strain which accompanies these symptoms in youth. The Goetsch test is very much like the reaction of excitement.

Book Review

ETUDE SUR L'EPILEPSIE TRAUMATIQUE. DR. PIERRE BÉHAGUE, Médecin aide-major de 1re classe au Centre neurologique de la Salpêtrière. Travail du Service de la Clinique des maladies du systême nerveux à la Salpêtrière (Prof. Pierre Marie), Louis Arnette, Paris, 1919, 320 pages.

This unusually good thesis is from the "militarized" service of Pierre Marie at the Salpêtrière, where of 3,623 patients with head injuries, 439 are known to have developed epilepsy. These cases form the basis of the present study. Owing to admirable cooperation between the neurosurgical service and the neurological service, and to a rule requiring patients to report at the hospital from time to time after discharge, the observation of a large proportion of the patients was unusually complete. In other words, the enormous material was wisely utilized and the result is a most valuable composite picture of traumatic epilepsy. The work is divided into three parts: I. Immediate and transitory epilepsy. II. Established epilepsy (épilepsie durable). III. Late symptomatic epilepsy.

I. These convulsions, which may be local or generalized, are caused by concussion injury, by foreign bodies or by hemorrhage, and soon disappear, or status epilepticus develops with fatal outcome unless the hemorrhage is surgically arrested. A little later convulsions may be caused by septic process.

II. This group of convulsions is due to concussion, obvious injury of the brain or to extracranial trauma which interferes with the cerebral circulation (injury or ligation of large vessels of neck).

It is worthy of note that the author found no case of so-called reflex epilepsy due to injury of a peripheral nerve.

One of the most instructive showings of the analysis of these obviously traumatic cases was that only one-third of the patients had focal paroxysms—motor, sensory or special sense—while two-thirds had generalized convulsions of the idiopathic type. In four-fifths of the cases the injury was either of the parietal (55 per cent.) or frontal (26 per cent.) region, but a few were caused by basilar fracture with no injury of the vault, and a few by concussion with no fracture and very little external injury. The analysis includes the relation of location of the trauma to character of the convulsions, the most various of brain injuries, the period of latency and many other clinical features. Like other investigators, the author found that syphilis and alcoholism predispose to traumatic epilepsy, that preexisting epilepsy is aggravated by brain trauma and that traumatic epilepsy is aggravated by subsequent injury. Most of the wounds causing epilepsy were septic.

The author's opinion on surgical intervention in head wounds is interesting. Surgery of the cranium alone is unimportant. But, early or late, operation on membranes or brain necessarily increases the already existing damage and hence favors the birth of epilepsy. Also of interest is his opinion of cranioplasty. He is sure that it may cause epilepsy; he found 22 cases in 99 cranioplasties. In those already epileptic the operation increases the convulsions or has no influence. He is also opposed to operation for cerebral hernia.

The latent period is shorter in the jacksonian type. Danger of epilepsy is greatest in parietal wounds; frontal, occipital and temporal wounds follow in the order given. The danger increases with the size and inversely with the velocity of the projectile; with the depth of the wound and especially with the extent and number of surgical procedures and the presence of suppuration.

The clinical descriptions are excellent, from prodromes and auras through different types of seizures to various epileptic equivalents. Diagnosis is adequately covered. There are twelve pages on pathogenesis with the following rather sterile conclusions:

1. Epilepsy is always caused by cortical irritation. This can be caused by a cicatrix, foreign body, tumor, etc., but especially by neuroglia cicatricial proliferation of hemorrhagic or traumatic origin.
2. The irritation is caused principally by expansion of the brain against the irritating substance.
3. This expansion is the result of ventricular dilatation due to increase of arterial tension which is facilitated by the peculiar structure of the choroid plexus.
4. The hypertension of the cerebrospinal fluid is secondary and can cause only generalization of the convulsion.

Regarding surgical treatment, Béhague endorses the conclusions of the interallied surgical conference, January, 1918. In brief these are: Jacksonian epilepsy may be benefited by removal of a foreign body, splinters, etc. The operation should be limited to removal of cortical irritants and no search made for deep seated bodies. Operations for other types of epilepsy are condemned. In medical treatment nothing new is offered.

III. By late symptomatic epilepsy the author means convulsions coming on after the head wound has healed, as a result of some complication, such as abscess, hemorrhage or nonsuppurative encephalitis. Such convulsions appear suddenly, are frequent and violent and demand immediate attention. An excellent microscopic study of two cases of nonsuppurative encephalitis closes this section.

The monograph concludes with notes of 444 cases, 13 formal conclusions and 22 pages of bibliography. It is a fine piece of work, valuable to neurologist and surgeon alike.

The American Medical Association will pay 50c each for the April and May, 1919, issues of the Archives of Neurology and Psychiatry. Address to American Medical Association, 535 North Dearborn St., Chicago, Ill.

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THE STATIC* AND KINETIC SYSTEMS OF MOTILITY

PRESIDENTIAL ADDRESS

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NEW YORK

The subject of motility is one of great interest to scientific men in many fields of investigation. Neurologists, particularly, have always had a peculiar interest in motor function and its disorders, and to neuropathology must be credited much that we know of the underlying neural and muscular mechanisms.

During the last half century our knowledge of this important function of the living organism has made great progress and each decade has marked some new advance in the realm of motion, as well as in the sensory sphere. For these two great functions of the living organism are the pillars on which have been reared those marvellous mechanisms of the central nervous system, the phylogeny of which is the story of all organic life.

The problem of motion and sensation must, therefore, ever remain a living issue in the realm of science, as life itself traverses these two pathways side by side in the slow and imperceptible processes of evolution.

It would carry us too far afield at this time to record the achievements of the many investigators whose names are imperishably linked with the history of motility. This I shall hope to do later when my subject is presented in more extended form. I wish, however, to acknowledge having made free use of this vast accumulation of facts in the conception of the efferent nervous system which I am about to formulate.

This hypothesis is not concerned so much with the elusive intricacies of motility itself, as with the dual nature of its underlying systems.

COMPONENTS OF MOTILITY

I believe that motility consists of two components, each being represented throughout the entire efferent nervous system by separate neural mechanisms, which are mutually cooperative and yet physiologically and

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* Read at the Forty-Sixth Annual Meeting of the American Neurological Association, New York, June, 1920.

anatomically distinct. This is true not only of the segmental nervous system but also of the larger integrating and coordinating masses of the brain and cerebellum. Under normal conditions they are inseparable and act in unison, and the harmony of their relation is only disturbed by the dissociation of accident or disease.

One of these components of motility is the movement proper, which is subserved by the *kinetic system.*

The other represents the more passive form of contractility, which we recognize in tonus, posture, attitude, and equilibrium. This static function of the efferent system is subserved by separate neuromuscular pathways, which may be termed the *static system.*

The term static is used here in the same sense as in the physical sciences, where it relates to the laws governing matter at rest and the forces producing equilibrium. I have found no other word which expresses better my conception of this other great function of the efferent system.

THE PHYLOGENY OF CONTRACTILE TISSUE

Before proceeding to a presentation of the evidence on which this conception is based, I would first ask your indulgence while I review briefly the phylogeny of the nervous system in its bearing on the development of movement.

All of those more complicated muscular activities which we study in man have, like every other function of the human organism, a very humble origin. And in order to detect the earliest manifestations of motility we must retrace our steps to the lowest rung of the ladder of life, where the ameboid movements of unicellular organisms allow these minute beings some slight increase in the range of their activities. Even in this lowly form the slow plastic movement is followed by a faint postural or static effort, when the pseudopodium of the cell becomes fixed to some neighboring object by molecular attraction or adhesion. The tiny organism then not only reaps the advantages of movement but the equally valuable effect of postural fixation under new conditions.

For if the power to move in space were not associated with an equal ability to control such movements in terms of posture, attitude and equilibrium, the advantages of motility would be of very doubtful value to the higher organisms.

The next advance in kinetic evolution is the ciliated movement. Here a protoplasmic cell has projecting from its free surface minute fibrils which are derived from the intracellular network of the organism. These cilia are capable of quick rhythmic, to and fro movements quite unlike the slow, plastic transformations of structure which characterize the first protoplasmic efforts of the ameba in the realm of movement.

As one ascends the scale of life, from one phylum to another, there is developed in the course of evolution a more complex contractile substance, the muscular tissue, which is the active agent in producing that great diversity of movement which characterizes animal life. The first development is the unstriped muscle which, according to some authorities, is present as low in the scale as sponges (Parker[1]), and is constant in all higher forms. The quality of its movement is slow and plastic. Striated muscle, which represents a higher differentiation of contractile tissue, makes its appearance in worms and mollusks, and constitutes the skeletal musculature of vertebrates. The quality of its movement is quick and elastic.

A very striking illustration of the dual nature of muscle function may be observed in certain of the invertebrates. In bivalved mollusks closure of the shell is affected by the contraction of two distinct muscles, a contractile muscle which is striated, and an arrest muscle which is nonstriated. The rapid movement of closure of the shell is produced by the striated muscle. This is accompanied by a slower contraction of the arrest muscle which becomes firmly fixed as soon as the movement ceases. The arrest muscle thus fixes in posture the closing movement of the shell, which is then able to withstand an enormous degree of pressure before relaxation is induced. A similar division of movement and posturing muscles may also be observed in Echinus and Pecten and these have separate and distinct sources of innervation (von Uexküll[2]).

Comparative anatomy shows clearly that the muscular system, like all other structures of the body, is in a state of evolution, adapting itself to the changing conditions of the organism. Development is from the slow movement of the unstriped to the quick movement of the striated muscle fiber. And in man all gradations may be observed from the lowest type of unstriped muscle to the highest type of striated muscle fiber.

Striated muscle contains two substances, both capable of contraction. The one is the disk mechanism of the muscle fiber, which gives rise to the quick movement or the twitch. The other is the sarcoplasm which yields a more plastic form of contraction. It is, I believe, not unlikely that the simple protoplasmic movements of the ameba and the quick rhythmic waves of the cilia are the prototypes of the more elaborate functions of striated muscle.

Marshall,[3] after an exhaustive study of the muscles of vertebrate and invertebrate life, has reached certain important conclusions. He believes that the disk mechanism of the muscle fiber is the final product in course of evolution from the primitive intracellular network of the cell. This is represented in the unstriped muscle fiber by the longitudinal fibrillations. He traces from its origin the gradual development of the contractile mechanism and shows its relation to the quality of movement. A movement is slow or quick according to the quality and degree of differentiation of this constituent of the muscle fiber.

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[1] Parker: The Reaction of Sponges, J. Exper. Zool. **8**:39, 1910.

[2] von Uexküll: Studien über den Tonus, Ztschr. f. Biol. **44**:269, 1903; ibid. **58**:305, 1912.

[3] Marshall, C. F.: The Structure and Distribution of Striped and Unstriped Muscle in the Animal Kingdom and a Theory of Muscular Contraction, Quart. J. Microsc. Sc. **27**:75, 1888.

A clear conception of the phylogeny of contractile tissue is important to my theory as I do not regard the skeletal muscle mass in man as a mere aggregation of muscle fibers of a standard type, but rather as systems of end organs representing different stages of phylogenic development, and thus corresponding in some measure to similar differentiation in nerve tissue.

I believe that the highest functions of motility are carried out by more highly developed muscle fibers than are the lower forms of movement. For the functions of the efferent nervous system and the muscular system are so closely intertwined that one can scarcely conceive of evolutionary progress in one without corresponding changes in the other, to meet the demands of more elaborate functional activity.

TYPES OF MOTILITY AND POSTURE

Let us now leave the question of muscle tissue and consider, for a moment, the general types of motility in animal life. These are usually divided by physiologists into three groups, reflex, automatic and associated, and isolated synergic movements.

It should be remarked, however, that one group merges imperceptibly into the other and the transition is so gradual that it is difficult to say where one type ceases and the other begins. For as Herbert Spencer pointed out, the progress of evolution is from the most automatic to the least automatic and cessation of automatic activity and the dawn of volition are one and the same thing.

In the lowest forms of life, movement and posture are of a purely reflex character and depend on a primitive reflex arc, which is the functional unit of the nervous system. In higher forms these reflex units are combined and more complicated reflex activities result. These in turn are gradually brought under the control of still higher integrating and correlating centers with corresponding advances in the variety and forms of movement.

There are many extraordinary evidences of reflex action in the lower forms of life, both in the realm of posture and of movement. There are such movements as the reflex of defense, the flight reflex, the turning reflex, that strange reflex of self-mutilation by which an extremity is discarded to escape a threatened danger. There is a disguise reflex characterized by alteration of color, also a tonic reflex producing a general rigidity and one simulating the posture of death, and many others that have been acquired in the struggle for existence.

In the "automatic and associated group" there are movements of crawling, walking, running, jumping, swimming and flying. These movements are usually bilateral and rhythmic, or alternating and rhythmic, and find their highest expression in vertebrates. All of these movements, reflex as well as automatic, have their corresponding static components, and every cessation of movement is immediately followed by fixation of the contraction in posture.

In the higher mammals and especially in man one encounters a new form of movement which differs from reflex and automatic movements in the quality of individuality and dissociation. Thus, one of the extremities which in reflex and automatic movements plays a fixed rôle as part of a mechanism, becomes capable of isolated and unilateral effort under voluntary control. This form of movement represents an immense advance in the evolution of motility and underlies the development of all skilled movements which have reached such extraordinary perfection in man.

In all of these isolated forms of voluntary movement there is also the element of posture, and it may be stated without fear of contradiction that there is no form of motility from the simple reflex to the most skilled and individualistic type of movement which functions without a corresponding static mechanism.

EVOLUTION OF THE NERVOUS SYSTEM

With the gradual increase in the range of movement and posture in organic life there has been a corresponding development of nerve structure, for every movement is represented as such in the mechanism of the nervous system. As one ascends the animal scale there is an increasing complexity in the arrangement of the central nervous system, due to the development of higher and higher levels of coordination and control. In man and the primates the original simplicity of the segmental neuraxis is quite concealed by the enormous development of cerebrum, cerebellum, and their respective projection systems in the spinal cord.

Hughlings Jackson,[4] following the philosophic teachings of Herbert Spencer, was the first to grasp the full significance of evolutionary principles as applied to the central nervous system of man. His conception of functional levels, each higher level controlling the activities of the lower centers, stands today, and will, to my mind, continue to dominate all conceptions of nerve functions, psychic as well as somatic. For in the central nervous system of man is written in terms of functional levels the whole history of the evolution of organic life.

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[4] Jackson, Hughlings: Croonian Lectures, Brit. M. J. **1**:660, 703, 1884.

In a general way the central nervous system may be said to represent three great structural and functional divisions. There is a segmental nervous system, which contains the great reflex systems of the neuraxis, a paleo-encephalon which represents the essential sensory and motor mechanism in lower forms, and a neo-encephalon which has its greatest development in man, and subserves the highest functions of motility and sensibility.

It is my belief that these three great functional divisions of the nervous system are related to the three great types of movement and posture enumerated in the foregoing.

In this conception, I regard the cerebellum as the great correlating center for the static functions of motility, in contrast to the corpus striatum and Rolandic area, which I regard as the essential correlating centers for the control of their respective forms of kinetic function. Furthermore, I believe that these systems are represented by special end organs in the skeletal muscles in the same manner as we conceive sensations with their special systems and modes of termination.

THE AUTHOR'S HYPOTHESIS

I will now proceed to a presentation of the evidence on which my hypothesis is based, which, however, on the present occasion, cannot be given in detail but can only be outlined in a very general way. Let us first consider the muscular system, which contains all of the contractile end organs of the efferent mechanism.

The skeletal musculature is composed of striated muscle fiber held together by connective tissue, by means of which the contractile mass performs its functions. The purely anatomic divisions of muscles are of comparatively little importance in the physiology of motion. For as Hughlings Jackson pointed out, "movements not muscles" are represented in the nerve centers and usually a number of muscles participate in any given movement.

We must, therefore, conceive the efferent nervous system, including the muscles, as organized for the execution of a great variety of movements which become more and more numerous and complex as we ascend the scale of life.

As we have seen, each striated muscle fiber is composed of two distinct substances which are now generally recognized by physiologists as subserving different types of motor function. In a general way it is estimated that the disk system constitutes about from one sixth to one half of the total muscle mass, the remaining portion of which is sarcoplasm.

DUAL INNERVATION OF MUSCLE FIBERS

Each muscle fiber contains a motor nerve ending, which is the terminal of a medullated nerve fiber. For many years this was thought to be the sole innervation of the muscle fiber, until Perroncito[5] and Boeke[6] demonstrated the existence of another smaller nerve ending in the striated muscle fiber which was shown to be the terminal of a nonmedullated nerve.

Boeke expressed the opinion that the motor end plate of the medullated nerve fiber furnished the stimulus for the disk system while the accessory sympathetic terminals were in control of the sarcoplasmic portion of the contractile mechanism, a conception which Mosso had previously expressed in his interpretation of the tonus and twitch components of muscle function. Boeke carried his investigation still further, and in order to prove the independent nature of these two systems, he cut the trochlear nerve in the cat and studied the nerve degenerations in the superior oblique muscle. All of the motor end plates in connection with the medullated nerve fibers were found in various stages of disintegration while the accessory sympathetic nerve endings showed no evidences of degeneration.

One could hardly offer better evidence of the independence of these two systems of nerve endings.

These histologic studies awakened considerable interest in the physiologic world, as Botazzi[7] had previously enunciated a theory of dual contractility for the muscle of the batrachian heart. In this hypothesis the slow tonic contraction of the sarcoplasm supported and reinforced the quicker movement of the disc system.

It is also interesting that physiologists have detected certain differences in the metabolism of muscles which are probably related to these two forms of activity of the muscle fiber.

Pekelharing[8] demonstrated the existence of two chemical processes in muscle metabolism. One was related to the disk system, and was characterized by the utilization of nonnitrogenous substances. The other was concerned with the utilization of albuminates and this was possibly dependent on the chemical activity of sarcoplasm. Other experiments have shown that the expenditure of energy of the active muscle contraction (tetanus) is far greater than is that of posturing muscle (tonus). This explains the relative unfatigability of the posturing or sarcoplasmic function of muscle. It is certainly significant that muscle fibers consisting of two types of contractile substance should also present two distinct forms of chemical activity.

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[5] Perroncito: Etudes ultérieures sur la terminaison des nerfs dans les muscles à fibres striées, Arch. ital. de Biol. **38**:393, 1902.

[6] Boeke: Die motorische Endplatte bei den höheren Vertebraten, Anat. Anzeiger **25**:193, 481, 1909.

[7] Botazzi: The Oscillations of the Auricular Tonus in the Batrachian Heart with a Theory on the Function of Sarcoplasm in Muscular Tissues, J. Physiol. **21**:1, 1897.

[8] Pekelharing: Die Kreatininausscheidung beim Menschen under dem Einfluss von Muskeltonus, Ztschr. f. physiol. Chem. **75**:207, 1911.

Another striking confirmation of a dual efferent system is to be found in the researches of Kure, Hiramatsu and Naito,[9] on the innervation of the diaphragm. These investigators were able to show by animal experimentation that the phrenic nerve conveys the kinetic impulses for this important muscle, while its tonus is received through the splanchnic system of the sympathetic. If the phrenic nerve is cut, the diaphragm is paralyzed but tonus is preserved. When the sympathetic nerve is cut, tonus is lost but motility is unaffected. As the diaphragm performs a relatively simple function it is not surprising that these two systems are dissociated and, as had already been shown by experiments of Boeke, the same is true for the kinetic and static systems of the ocular muscles..

The attempt has also been made to demonstrate a similar dissociation in the innervation of the lower extremities by cutting the trunk of the abdominal sympathetic. This experiment was performed by Dusser de Barenne[10] who claims to have produced a demonstrable loss of tonus in the corresponding muscles. These results, however, have not been confirmed by subsequent observers (Cobb,[11] Beritoff,[12] and others) and it appears probable that the sympathetic efferent system of the extremities takes a different route, not passing by way of the vascular system.

As the extremities represent a later phyletic addition to the muscular system, it is possible that the efferent static innervation for these structures may pass to the muscles with the motor nerves. In this connection it is interesting to note that Ransom[13] has shown the existence of a large complement of nonmedullated nerve fibers in peripheral nerves. Further investigation will be necessary, however, to clarify this question and to show any relationship between the nonmedullated fibers described by Ransom and the sympathetic terminals of the striated muscle fiber.

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[9] Kure, Hiramatsu and Naito: Zwerchfelltonus und Nervi splanchnici, Ztschr. f. exper. path. und Therap. **16**:395, 1914.

[10] Dusser de Barenne: Ueber die Enthirnungsstarre (Decerebrate Rigidity) in ihrer Beziehung zur efferenten Innervation der quergestreiften Muskulatur, Folia neuro-biol. **7**:651, 1913.

[11] Cobb: A Note on the Supposed Relation of the Sympathetic Nerves to Decerebrate Rigidity, Muscle Tone, and Tendon Reflex, Am. J. Physiol. **46**:478, 1918.

[12] Beritoff: Die tonische Innervation der Skelettmuskulatur und der Sympathicus, Folio neuro-biol. **8**:421, 1914.

[13] Ransom: Unmyelinated Nerve Fibers as Conductors of Protopathic Sensation, Brain **38**:381, 1915.

The existence of two separate contractile systems in striated muscle fiber is also shown very clearly by the electromyogram. In myotonia congenita, Gregor and Schilder[14] have demonstrated the presence of two distinct contraction waves, the one quick and referable to the disk system, the other slower, more sustained, and of sarcoplasmic origin. A similar dissociation of quick and slow muscle waves may also be produced in the electromyogram of veratrin and creatinin intoxication.

PERIPHERAL MECHANISMS OF TWO SYSTEMS OF MOTILITY

In the theory which I am endeavoring to unfold the disk is to be regarded as the end organ of the kinetic system, and the sarcoplasm as the end organ of the static system, just as we conceive the heat and cold spots or the tactile corpuscles as end organs in their respective sensory spheres. These end organs of the skin have undergone special differentiation on certain parts, and have thus acquired a greater accuracy and refinement of function.

I believe that a similar specialization of function and structure exists in skeletal muscles. By this I would emphasize what perhaps is not generally recognized, that the muscle fiber system, like the nerve fiber system, participates in phyletic differentiation, and that those muscle fibers which subserve the higher functions of neokinesis are more highly specialized than are those which are concerned with paleokinetic function.

According to this conception the corticospinal system and the striospinal system would both be represented by special contractile end organs.

As the striospinal system is older, the muscle fibers subserving this function would be less highly differentiated than those subserving the highest cortical function. Let us see what facts may be derived from histology, and physiology, in support of this point of view. As long ago as 1874, Ranvier pointed out that in muscles there are two kinds of fibers, which he described as pale and dark fibers. The pale fibers are rich in contractile substance and contain comparatively little sarcoplasm. The dark fibers are rich in sarcoplasm with a relatively simple disk mechanism. Both kinds of fibers are found in all muscles in varying degrees.

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[14] Gregor and Schilder: Zur Theorie der Myotonia, Neurol. Centralbl. **32**:85, 1913.

The pale fibers are the quick contracting, and the dark fibers the slow contracting fibers of the physiologists (Pankúl[15]). And the quickness of a muscle contraction which varies considerably in different portions of the body depends on the relative number and distribution of these two types of fibers. It has also been shown that those muscles which are subjected during the life of the organism to the most constant activity are especially rich in sarcoplasm (Knoll[16]).

I believe, therefore, that the more highly differentiated pale fibers are related to the neokinetic system, while the dark fibers are concerned with paleokinetic motility. As the evolution from the lower to the higher movement is gradual, there are transition forms between these two types of fibers.

The skeletal muscles may therefore be regarded as contractile tissue, which bears the imprint of the phylogenetic struggle in the same measure as the nervous system which controls it. Considered as a whole it is the end organ of the efferent system and in its mass are represented the great kinetic and static pathways of the brain and cord.

THE SEGMENTAL NERVOUS SYSTEM AND THE DUAL FUNCTION OF MOTILITY

Let us now consider the segmental nervous system in its relation to a dual function of motility. At this level are represented the earliest centers for movement and posture, and the relative simplicity of its reflex mechanisms has made it an especially attractive field for experimental study. Sherrington,[17] who is the acknowledged authority on the various reflex activities of the nervous system, has reached the conclusion that two types of reflex action may be recognized. There is a reflex movement which he calls the phasic reflex, and a reflex posture which he terms the tonic or postural reflex, and in the realm of reflex action he indicates very clearly the duality of function which I would extend to the whole of the efferent system.

He also has shown in his study of "postural activity of nerve and muscle" that coordination and reciprocal innervation are as much a part of the tonic, or postural, reflex as they are of the phasic reflex mechanism. His experiments on decerebrate rigidity, as well as those of Magnus and Kleijn,[18] show conclusively that innervation and coordination are as important in the realm of posture as in that of movement.

Reflex action is therefore dependent on a peripheral kinetic system which terminates in the disk contractile mechanism. And reflex posture

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[15] Pankul, in Luciani: Textbook of Physiology **3**: Chapter 1.

[16] Knoll: Ueber Protoplasmaarme und Protoplasmareiche Muskulatur, Denkschr. d. k. Akad. d. Wiss. z. Wien. Math. naturw. Kl. **58**:633, 1891.

[17] Sherrington: Postural Activity of Müscle and Nerve, Brain **38**:191, 1915.

[18] Magnus and Kleijn: Die Abhängigkeit des Tonus der Extremitäten muskeln von der Kopfstellung, Pflüger's Arch. f. d. ges. Physiol **145**:455, 1912.

is dependent on a peripheral static system which terminates in the sarcoplasm of the muscle fiber. These are the neuromuscular representatives at the segmental level of the kinetic and static pathways of motility.

One function of the peripheral static system is the so-called "lengthening and shortening reactions" of muscles. This intrinsic function of muscle tissue is dependent on postural tonus, by which a muscle may be lengthened or shortened during contraction without corresponding changes of tension. This alteration in postural length without corresponding changes of tension facilitates the easy transition from posture to movement which characterizes motility.

Sherrington has also shown the great importance of this posturing mechanism in hollow viscera, like the bladder, the postural tonus of the muscular walls of this organ allowing considerable changes of volume without increase of the intravesical tension. It has also been definitely established that a similar mechanism exists in the vascular system and in other structures under the control of the autonomic nervous system.

Before leaving the peripheral systems of motility, some mention should be made of the "all or nothing" principle of Bowditch [19] which apparently governs all forms of muscular contraction. His experiments were carried out on the heart muscle of the frog and showed that an induction shock produces a muscle contraction or fails to do so according to the strength of the current. If a contraction is induced, it is the greatest of which the muscle fiber is capable, and there are no gradations with varying strengths of current.

Keith Lucas [20] has shown that this principle also applies to the skeletal muscles. Accordingly, the strength of a muscular contraction is not dependent on the strength of the current, but on the number of muscle fibers participating in the contraction. The refinements and gradations of movement are therefore dependent on the number of muscle fibers participating, and not on variations in the strength of the nerve impulse.

CENTRAL PATHWAYS OF TWO SYSTEMS OF MOTILITY

Let us now leave the peripheral mechanisms and pass to a consideration of the central pathways of motility.

In a previous study of paralysis agitans,[21] I expressed myself as favoring the conception of two great motor systems from brain to muscle, one subserving paleokinetic and the other a neokinetic function. The corpus striatum, according to this view, is the paleokinetic center for the control of "automatic and associated movements," and the Rolandic area is the neokinetic center for "dissociated movements" of cortical origin. Their static functions I believe are subserved by other pathways.

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[19] Bowditch, in Bayliss: Textbook of Physiology, 1919, p. 383.

[20] Lucas, Keith, in Bayliss: Textbook of Physiology, 1919, p. 383.

[21] Hunt: Primary Atrophy of the Pallidal System: A Contribution to the Functions of the Corpus Striatum, Brain **40**:58, 1917; Arch. Int. Med. **22**:647, 1918.

It should be remarked, however, that the thalamic portion of the paleo-encephalon is closely associated with the cerebral cortex by afferent and efferent tracts, by means of which paleokinetic motility may be directly controlled from the cortical sphere (cortico-thalamo-striospinal system). This higher control of the striospinal system makes possible a finer differentiation of paleokinetic motility and explains the occurrence of purely involuntary movements of cortical origin. It also bridges the gap between the striospinal and corticospinal types of movement and renders the transition from the older to the newer forms of motility a more gradual process.

In striking contrast to the paleokinetic system is the neokinetic system. This is a direct pathway from the cerebral cortex to the spinal cord for the control of isolated synergic movement. It takes its origin in the motor cells of the Rolandic area and passes by way of the pyramidal system directly to the anterior horn cells of the spinal cord, and from there to a special disk representation in muscle subserving the dissociated movements of cortical origin.

These two great motor systems, the pyramidal and extrapyramidal, according to this theory, are both represented in nerves and skeletal muscles. They are in a sense complementary and their independence is only unmasked by the dissociations of disease.

A critical analysis of the symptomatology of pyramidal and extrapyramidal disease (spastic paralysis and paralysis agitans) are strongly corroborative of this hypothesis (Hunt[22]).

The central pathways for the control of the static component of motility are, I believe, distinct from those subserving its kinetic function. The essential suprasegmental mechanism for the control of static motility I believe to be the cerebellum. The importance of this organ in the regulation of muscle synergy and tonus and in the maintenance of equilibrium has been recognized by all observers, and yet, notwithstanding years of patient investigation, a certain obscurity still clouds our conception of its function and relation to motility.

DeBoer[23] has recently considered it as the central mechanism for the control of reflex tonus and the sarcoplasmic function of the muscle fiber, and has presented an imposing array of evidence in favor of this hypothesis. Tilney,[24] in a comprehensive study of the cerebellum, has enlarged our conception of its synergic function to include the synergic unit. This is represented by a single muscle and its antagonist which contains a "dominant and check mechanism" capable of kinetic and akinetic phases. In this conception both the kinetic and static functions are subserved by the unit of synergy.

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[22] Hunt: The Existence of Two Distinct Physiological Systems for the Transmission of Motor Impulses in Peripheral Nerves, Brain **41**:302, 1918.

[23] de Boer: Die Bedeutung der tonischen Innervation für die Funktion der quergestreiften Muskeln, Ztschr. f. Biol. **47**:239, 1914.

While accepting in the main the classic conception of cerebellar synergy as enunciated by Babinski, I would limit this function to a control of the static synergies of motility. The kinetic synergies are controlled by their respective kinetic mechanisms.

Anatomically, the cerebellum has close connections with both the cerebrum and the spinal cord. It receives impulses directly from the spinal cord, which pass to its central portion (vermis), as well as impulses from the cerebral cortex, which pass by way of the pons varolii to the cerebellar hemispheres.

In the cerebellum there are phylogenetic evidences of two great systems, which correspond to similar divisions of the cerebrum. There are a paleocerebellum, or vermian system, and a neocerebellum or hemispheric system.

The efferent cerebellar systems originate in the central nuclei (the dentate, nucleus globosus, emboliformis and fastigii) and traverse the superior cerebellar peduncles.

The older cerebellum stands in relation to what may be termed the *paleo-static system* which controls the static functions of movement of the "automatic and associated type." It takes its origin in the older nuclei of the vermis cerebelli.

The cerebellar hemispheres, on the other hand, regulate the higher postural functions of motility through the medium of a *neo-static system*; which originates in the cells of the dentate nucleus. This is controlled from the cerebral cortex by a special system of fibers passing from the frontal lobe to the pons varolii and thence to the opposite hemisphere of the cerebellum. The frontopontocerebellar system descends in the internal capsule and cerebral peduncle in close association with the pyramidal tracts, and it is interesting to note that both structures, the one subserving the static and the other the kinetic function of motility, receive their myelin sheaths subsequent to birth.

Both of the efferent systems of the cerebellum pass in the superior peduncles to special cellular divisions of the nucleus ruber (nucleus magnocellulatus and nucleus parvicellulatus), from which they descend as separate systems in the spinal cord, a paleorubrospinal system and a neorubrospinal system. I then conceive them as passing in separate systems to the sarcoplasm of the skeletal muscles.

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[24] Tilney: Functional Significance and Principal Syndrome of the Cerebellum, Neurol. Bull. **2**:289, 1919.

Before leaving the question of the central pathways, I would say a word in respect to a group of subordinate motor centers which are found scattered in the tegmental region of the brain stem, and usually referred to as prespinal centers of motility.

The prespinal or better supraspinal centers of motility represent a distinctly lower level of phylogenesis than the corpus striatum. They are intercalated in the pathway of the extrapyramidal system, of which they form a part and I believe contain representatives for both the kinetic and the static components of motility. With certain exceptions these cannot be differentiated in the present state of our knowledge. The red nucleus, however, may be cited as an example of a purely static center at the supraspinal level of motility. The manifestations of decerebrate rigidity are probably closely related to this supraspinal static mechanism.

MOTOR MANIFESTATIONS OF DISEASE

I would now pass to a consideration of the efferent system in its relation to symptomatology. Is it possible to recognize the existence of a static and kinetic system in the various motor manifestations of disease?

In the interpretation of such phenomena one should always consider the existence of positive and negative symptoms as differentiated by Hughlings Jackson.[25] The positive symptom occurs in lower centers from loss of the inhibitory action of higher centers. Many important motor disturbances are of this order and are referable to secondary functional discharge in lower centers.

In a general way, it may be said that a lesion of the kinetic system causes a disorder of movement, and a lesion of the static system, a disorder of tonus or of posture.

In many of the disturbances of motility both systems participate, although it is usually possible to indicate one or the other as the essential factor involved.

At the segmental level of the nervous system the tendon reflex is a typical example of kinetic function, as is the muscle tonus of static function. Langelaan [26] has recently shown that tonus consists of two components, a contractile tonus referable to the disk system and a plastic tonus of sarcoplasmic origin. These may also be recognized on direct percussion of the muscle by the quick contractions of myotatic irritability and the more persistent local reactions of the idio-muscular response.

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[25] Jackson, Hughlings: The Evolution and Dissolution of the Nervous System, Brit. M. J. **1**:591, 1884.

[26] Langelaan: On Muscle Tonus, Brain **38**:235, 1915.

A similar division of function is also demonstrable in reflex action, the kinetic system yielding reflex movements, and the static system reflex posture.

Such disorders of motility as paramyoclonus multiplex, myokymia, and fibrillary twitchings are spasmodic phenomena referable to the kinetic spinal mechanism, while myasthenia gravis is a paretic manifestation. The clonus of spastic paralysis and the tremor of paralysis agitans are both of kinetic origin. One is related to the neokinetic, and the other to the paleokinetic system. The more plastic tonicity which is also present in both of these forms of palsy is of sarcoplasmic origin and referable to the static system. Certain well marked differences in hypertonicity which characterize the two disorders I would refer to difference of static and kinetic representation in the skeletal muscle. Huntington's chorea and epilepsy are both of kinetic origin, the former is referable to the striatal and the latter to the cortical level of motility.

Other disorders of motility, referable to the corpus striatum, such as athetosis, dystonia, progressive lenticular degeneration and paralysis agitans, represent different degrees of involvement of its efferent and inhibitory systems. This phase of the subject has been considered in detail in my contributions to the symptomatology of the corpus striatum.[27]

Rarely, in epilepsy there are seizures which suggest a relation to the static system. These are characterized by unconsciousness and a sudden loss of postural control without convulsive manifestations (static epilepsy).

All forms of myotonia, cerebral, cerebellar, spinal and peripheral, would appear to be of sarcoplasmic origin and referable to the static system. There is also much in favor of the hypothesis that the tonic spasms of cerebellar origin and the tonic rigidity of tetanus are essentially contractile manifestations of sarcoplasm.

The nature of the Bárány reactions to labyrinthine stimulation also indicate, in my opinion, a relationship to static function and the sarcoplasm of muscle. I would recall the slow and quick components of labyrinthine nystagmus. The slow movement is primary and referable to excitation in the static sphere, while the quick movements are secondary, and of kinetic origin.

The relationship of the frontopontocerebellar system to various myotonic phenomena has already been emphasized by Kleist [28] and Kinnier Wilson. Mills [29] has also considered this question in a discussion of a cerebral tonectic mechanism. Kinnier Wilson and Walshe [30] have made the important observation that tumors of the frontal lobe anterior to the motor area may produce myotonia, a peculiar feature of which is the limitation of the disturbance to voluntary movements. Bumke [31] has also described a family disorder characterized by the occurrence of myotonia with both voluntary and automatic type of movement. It should be remarked that in all of these central forms of myotonia the usual electrical and mechanical evidences of myotonia congenita are absent, which proves the disorder to be of central origin.

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[27] Hunt: The Efferent Pallidal System of the Corpus Striatum, a Consideration of Its Functions and Symptomatology, J. Nerv. & Ment. Dis. **46**:211, 1917.

[28] Kleist: Ueber nachdauernde Muskelkontraktionen, J. f. Psych. u. Nerv. **10**:95, 1908.

[29] Mills: Muscle Tonicity, Emotional Expression and the Cerebral Tonectic Apparatus, Neurol. Centralbl. **33**:1266, 1914.

These various clinical types of myotonia I regard as corroborative of the conception of a special system for the control of the higher function of static motility.

There is one organic symptom in particular which is closely related to the static function of the cerebellum, and that is the intention tremor. The intention or cerebellar tremor presents the characteristics of a loss of the static components of motility. The coarse tremor movements may be regarded as an effort of the kinetic system to compensate for the loss of the posturing or static functions of the sarcoplasm.

In a movement complex, the kinetic component governs the synergies of the movement itself, but is accompanied by the static function which guides, steadies and reinforces the movement in its course and fixes it in posture when the movement ceases. When this mechanism is disturbed the intention tremor results.

At a meeting of this Association five years ago, I [32] described Dyssynergia cerebellaris progressiva, an affection characterized by the gradual development of generalized intention tremors. On the basis of pathologic investigations not yet reported, I would refer this disorder to an atrophy of the efferent dentate system through which the cerebellum exercises control of the static system of motility (primary atrophy of the efferent dentate system).

In addition to these various somatic expressions of kinetic and static function, a similar division may be recognized in the mental sphere. Among these may be mentioned catalepsy, catatonia, and certain of the hyperkineses of psychic origin. A psychostatic and psychokinetic representation may therefore be postulated at the psychic level of motility.

This concludes my argument in favor of the two great systems of motility; it is only an outline, and were my time not limited it could be reinforced by many additional facts which would materially strengthen the hypothesis.

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30. Wilson and Walshe: The Phenomena of Tonic Innervation and Its Relation to Motor Apraxia, Brain **37:**119, 1914.

31 Bumke: Eine familiäre Form von Intentionskrampf Ztschr. f. d. Neurol. u. Psych. **4:**644, 1910.

32. Hunt: Dyssynergia Cerebellaris Progressiva, Brain **37:**247, 1914.

SUMMARY OF HYPOTHESIS

In conclusion, I would restate briefly the general principles underlying this conception of the efferent system. The function of motility, like sensation, is represented by special end organs and neural systems which subserve the various types and modalities of contractile function. Their phylogenesis is in strict accord with the evolutionary hypothesis. From the primitive movement and posture of the lower forms of life to the highest expressions of motility in man, the transition is a gradual one and is recorded in the efferent nervous system of man.

As the animal organism is subject to the mechanical laws of a physical world, two forms of movement, active and passive, have been evolved in the struggle for existence. The one is that passive contractility of the muscular system which maintains the organism in posture and equilibrium and counteracts the forces of gravity and atmospheric pressure. The other is a more active form of contractility by which the organism is enabled to move or change its position in space.

In some invertebrates these two forms of motility are readily distinguished and are subserved by separate muscles. In higher forms these two important functions of muscle tissue are united in the muscle fiber, but are controlled by separate neural systems.

It is therefore possible to trace the evolution of the static and kinetic systems of motility from the lowest to the highest levels of the neural mechanism.

ACQUIRED DOUBLE ATHETOSIS *

(DYSTONIA LENTICULARIS)

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In 1916 I [1] reported several cases of a disorder occurring in one family, which seemed to me to belong to the group known as pseudosclerosis, probably a family form of a lesion of the lenticular nucleus, and in that paper I grouped the various disorders attributed to disease of the lenticular nucleus. These were the pseudosclerosis of Westphal and Strümpell, Huntington's chorea, Parkinson's disease, spastic pseudobulbar palsy with contractures and choreo-athetoid movemeünts of Oppenheim and Vogt, and Freund and Vogt, Oppenheim's dystonia musculorum deformans, and double athetosis. To this list I added von Bechterew's hemitonia apoplectica and certain forms of carbon monoxid poisoning, and I am inclined to add a certain form of paramyoclonus multiplex resulting from lethargic encephalitis. To the list might also be added arteriosclerotic muscular rigidity and certain forms of senile dementia, which Strümpell thinks belong to his amyostatic syndrome. He refers to a paper on the arteriosclerotic form of muscular rigidity by O. Förster, but is unwilling to attribute to arteriosclerosis of the brain arteries so great causal and primary significance as Förster has done. He thinks other etiologic factors are to be considered and that while arteriosclerosis is a frequent accompanying condition, it is not a conditio sine qua non.

Nonne says that numerous findings in lethargic encephalitis show that acute inflammation of the basal ganglions may cause the amyostatic syndrome. It seems to me that the involuntary jerkings of lethargic encephalitis probably have their origin in these structures, whether they assume a choreiform or myoclonic type. A few months ago a case of the myoclonic form of lethargic encephalitis with intense muscular contractions of the myoclonic type in various parts of the body occurred in my service and terminated fatally. I found in microscopic sections a pronounced cellular infiltration of the region of the basal ganglions, but not confined to them. It occurred diffusely in the basal part of the temporal lobe. The intense perivascular

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* Read before the Forty-Sixth Annual Meeting of the American Neurological Association, New York, June, 1920.

[1] Spiller, W. G.: Pseudosclerosis, J. Nerv. & Ment. Dis. **43**:23 (Jan.) 1916.

cellular infiltration of the region of the aqueduct of Sylvius, so common in the usual form of lethargic encephalitis, did not occur in this case.

REPORT OF A CASE

History.—M. A., a girl, aged 16, was admitted to the Philadelphia General Hospital, Sept. 5, 1882, and died there Dec. 24, 1919. The notes taken when she was admitted stated that her father was dead, but her mother, one brother and one sister were living and well. The patient's intelligence was said to be good. When she was about 5 years of age it was noticed that she had begun to drag her left lower limb and that her left upper limb was useless. Other parts of the body became successively affected and the paralysis assumed a spastic character, the head became drawn to the left, the body arched to the left, the knees flexed and the feet placed in the position of talipes varus. Marked incoordinate movements were observed in the hands and feet; the limbs on the left side became useless, but the right hand still had some voluntary function. She could not walk and could not sit in a chair unless tied in it. Articulation was difficult and defective.

Dr. George Wilson made some notes on Jan. 29, 1915. She had been in the Philadelphia General Hospital thirty-three years. She was irritable and had an exceedingly violent temper. She objected strenuously to being examined and on one occasion called the examiner an abusive name with unusual distinctness. She had a peculiar explosive speech, which was indistinct; occasionally words were said distinctly. One who associated with her constantly was able to understand what she said. She had difficulty in chewing and swallowing and had to be fed.

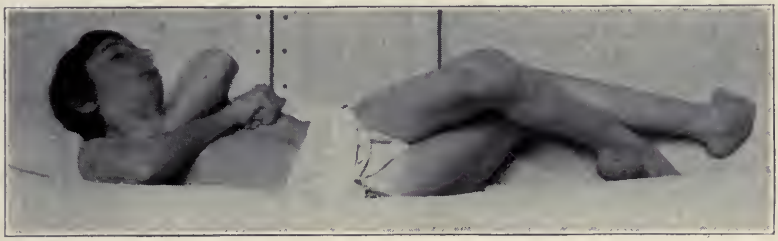

Fig. 1.—Spastic contractions with athetoid movements. These photographs were taken by Dr. Ralph Pemberton some years before the death of the patient.

Examination.—She appeared shrunken. Her irides seemed to react to light and in convergence. She had no nystagmus and no exophthalmos. Her lower jaw was frequently drawn downward and to the left and the movements were typically athetoid. Her upper limbs had little voluntary power. The fingers were tightly clasped into the palms and the forearms were supinated or pronated slowly and unevenly. The upper limbs were hypertonic and the tendon reflexes could not be obtained satisfactorily.

The lower limbs were extremely spastic and presented athetoid movements, the right foot was strongly inverted and the right great toe extended in the movements, while the left foot was placed so strongly in the position of equinus that the toes were more posterior than the heel. Plantar stimulation gave no response except athetoid movements, which seemed more pronounced when external stimulus was applied to the feet. The patellar reflexes were not obtained on account of the spasticity. At times when the athetoid movements relaxed the entire right lower limb seemed to go into clonus. The sensation of pain was preserved.

On Feb. 21, 1916, the condition was about the same. All the limbs showed contractures and there was some muscular atrophy. Mental deterioration was marked.

On June 10, 1916, it was noted that she agreeably cooperated in the examination to the best of her ability. While mentality was poor she showed no attacks of exaltation or depression, and no errors of sense perception or of judgment.

Her muscular system was continually in a universal athetosis. The arms, hands, legs and feet were most involved and to an extreme degree, the neck was less involved, and the trunk only slightly involved. There was a spastic scoliosis. The tendon reflexes were not obtained, possibly because of the spasticity. The muscles were moderately atrophied. Both pupils showed myosis. On Dec. 28, 1916, she had periodic attacks of diarrhea. She had myocarditis and her death on Dec. 24, 1919, was attributed to this cause.

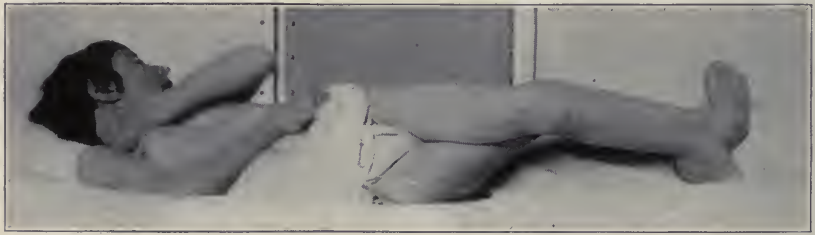

Fig. 2.—Spastic contractions with athetoid movements.

Necropsy Examination.—The necropsy examination was performed by Dr. Edward Weiss. The liver showed passive congestion; its weight was 510 gm. It was long and narrow, its surface was granular, the capsule was transparent and the substance finely mottled. It showed marked congestion of the central acini of the lobules with dilatation of the contributing sinusoids and atrophy and pigmentation of the surrounding liver cells, with slight increase in the supporting tissue. The diagnosis was chronic passive congestion and red atrophy.

The spleen showed passive congestion. It was small and firm; the interior was dark red; the trabeculae were well marked. It weighed 70 gm. It showed hyperplasia of the follicles, considerable increase in connective tissue supportive structure and splenic capsulitis. The diagnosis was chronic diffuse splenitis.

The left kidney weighed 80 gm.; the right 90 gm. They showed slight chronic interstitial nephritis. The left kidney was small and firm and cut with increased resistance; the capsule stripped with difficulty, leaving a finely granular surface; the cortex was narrow and the cortical striations distinct. The left suprarenal gland grossly was normal. The right kidney and suprarenal gland were about the same as the left.

The length of the body was 50 inches. The brain and cord weighed 1,170 gm. They were studied by me. The convolutions of the frontal lobe were distinctly smaller than those of the rest of the cerebrum, but there were no sclerotic areas in the cortex.

Microscopic Examination.—A horizontal section was made through the upper part of the basal ganglions. Each lenticular nucleus was about one-half the normal size. The globus pallidus on each side was firm but the putamen on each side had a worm eaten appearance and contained numerous small holes. A second horizontal section was made through the cerebrum about 1 cm. below the first section, and the basal ganglions from the left cerebral hemisphere in this upper piece were cut in serial sections. The basal ganglions on the right side were cut in serial section below the second cut. As the basal ganglions from the two sides had the same macroscopic and microscopic appearance, it was not considered necessary to cut all parts of them, especially as it was desirable to keep a portion of them intact on account of their gross appearance.

Sections from the tip of the right frontal lobe were stained with thionin to determine the cellular condition. They did not show any distinct pathologic condition. The Betz cells of the paracentral lobules were greatly altered, the nucleus was eccentric and the chromatolysis was pronounced, but the smaller nerve cells of the paracentral cortex did not appear to be greatly altered. Weigert hematoxylin sections of the paracentral lobules were normal.

The nerve cells of the left island of Reil were in good condition. Nowhere could an increase in size of the neuroglia nuclei be found. The left putamen was made up of shreds of tissue containing numerous neuroglia nuclei but only here and there a nerve cell, small and much altered, was found. The Weigert hematoxylin stain showed numerous separate, wavy, medullated fibers in the putamen, each fiber standing out distinctly on the brown background. The blood vessels of the putamen were much thickened and the perivascular spaces were much enlarged.

The globus pallidus was atrophic, as was also the nucleus caudatus, but neither showed the peculiar tissue seen in the putamen. The external and extreme capsules were well defined by the Weigert hematoxylin stain. The internal capsule, both the anterior and posterior limbs, was normal. The thalamus was about normal in size and appearance.

The putamen of the right side of the brain showed the same condition as that of the left side. The perivascular spaces within it were greatly enlarged. The globus pallidus, aside from a diminution in the number of medullated nerve fibers and a smaller size, was not greatly altered. The ansa lenticularis was probably a little smaller than normal. The cells of the nucleus ruber were in good condition and the nucleus was of normal size. The corpus dentatum appeared to be normal, but the cells were much pigmented. The anterior cerebellar peduncles were normal.

The nerve cells in the anterior horns of the spinal cord in the cervical and lumbar regions by the thionin stain appeared on the whole to be normal, except that here and there an intensely altered cell was found, in which the cell body was swollen, the nucleus was displaced and chromatolysis was pronounced. The pyramidal tract was normal throughout the brain and spinal cord.

DIFFERENTIAL DIAGNOSIS

Lenticular Degeneration.—When one reads the clinical description Wilson [2] gives of his progressive lenticular degeneration, the resemblance to bilateral acquired athetosis is striking. He says: It consists of involuntary movements, nearly always a bilateral tremor of both upper and lower extremities, the head and trunk also being sometimes involved—a tremor usually rhythmical but occasionally irregular, and increasing with volitional movement; there is pronounced spasticity of the limbs and of the face, the latter being usually set in a spastic smile, while in the later stages contracture of the limbs develops; there is dysphagia and dysarthria, the latter eventually degenerating into complete anarthria; there is sometimes spasmodic laughing and emotionalism. As a result of the extraordinary degree of stiffness of the musculature there is considerable difficulty in maintaining equilibrium. Little or no true paresis or paralysis occurs, however, as most ordinary movements, if not all, can be executed, though it may be slowly and feebly, and there is some weakness. In some cases certain mental symptoms, of a transitory nature, manifest themselves. There is no sensory disturbance and no alteration in the cutaneous reflexes. If the abdominal reflexes are absent (apart from muscular rigidity) or the plantary reflex is of

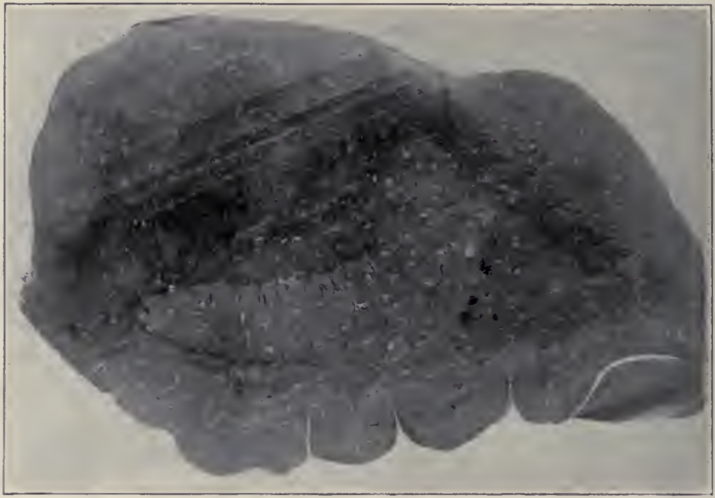

Fig. 3.—Normal section through the basal ganglions. Compare this with the section represented in Figure 4 taken at about the same level from the case of double athetosis.

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[2] Wilson: Brain **34**:295, 1912.

the extensor type, the syndrome is no longer pure. This, Wilson holds, is the clinical picture in pure, uncomplicated, bilateral lesions of the lenticular nucleus, and more generally of the corpus striatum, provided they are of sufficient size and of adequate duration.

Wilson says Gowers, Ormerod, Homen and he (Wilson) have been struck by the odd way in which the patients, seemingly so inarticulate, have occasionally been able to utter words or phrases with comparative ease and distinctness. This was especially observed in my case of double athetosis when, on one occasion, the patient said "damned fool" with great distinctness, whereas usually she could hardly be understood.

The lesions Wilson found in the lenticular nucleus in his cases consist of glial overgrowth which disintegrates and breaks down; there is often an immense increase in glial nuclei; the nerve fibers and nerve cells of the normal nucleus disappear; "Körnchenzellen" and macrophages are frequently present in numbers. Even when the cavity formation is extreme there are no signs of obliterative endarteritis in the blood vessels, the perforating lenticulostriate vessels and their branches. In advanced cases there are degeneration of the ansa lenticularis, relative atrophy of the corpus Luysii, partial degeneration of the lenticular bundle of Forel and of the strio-Luysian fibers and degeneration of striothalamic fibers. Apart from the degeneration in the lenticular nucleus, the changes in the brain are insignificant. Thus, the pathologic finding is bilateral symmetrical degeneration of the putamen and globus pallidus, especially of the former. The caudate nucleus is often somewhat atrophic, but never to the same extent. The Betz cells are intact and the cerebral cortex offers little definite alteration.

Although Wilson found no important cortical changes in his cases, others have reported such findings. It is impossible to explain all the symptoms of progressive lenticular degeneration by lesions of the lenticular nucleus, and this has impressed Strümpell, who states that the psychic symptoms (dementia, excitement and other symptoms) cannot be attributed to the disease of the lenticular nucleus.

In the case of progressive lenticular degeneration reported by Lhermitte[3] the findings in the lenticular nucleus closely resembled those in my case of athetosis. He found atrophy of the putamen and caudate nucleus, and the putamen contained lacunes of disintegration. The vessels were normal in these lesions. The picture he gives of the lesions resembles the findings of my case but the putamen appears less intensely affected. He says the nerve cells of all regions of the cerebral cortex were atrophied and full of lipochrome granules.

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[3] Lhermitte: Semaine méd. **32**:121 (March 13) 1912.

Schütte[4] reported a case which he thought resembled progressive lenticular degeneration, and he found decided alteration of the brain, especially of the frontal lobe, where the nerve elements were destroyed in places. The remaining cerebral cortex was diffusely affected but much less intensely. The basal ganglions showed some change in the ganglion cells and increase of neuroglia, but only in slight degree, and yet the symptoms were not essentially different from those of Wilson's cases. Schütte questions whether the lenticular nucleus is the seat of the disease. He also found the liver diseased.

In the case of progressive lenticular degeneration reported by Pfeiffer,[5] in addition to lenticular lesions the cerebral cortex was much altered. The ganglion cells in the frontal region were more severely affected than in other parts of the cortex. A swelling of the cells was observed, but shrinkage and other types of cellular disease were likewise frequently encountered, limited to the small and medium sized cells. In the motor region the giant cells were not perceptibly affected, but the commissural and associated neurons were altered.

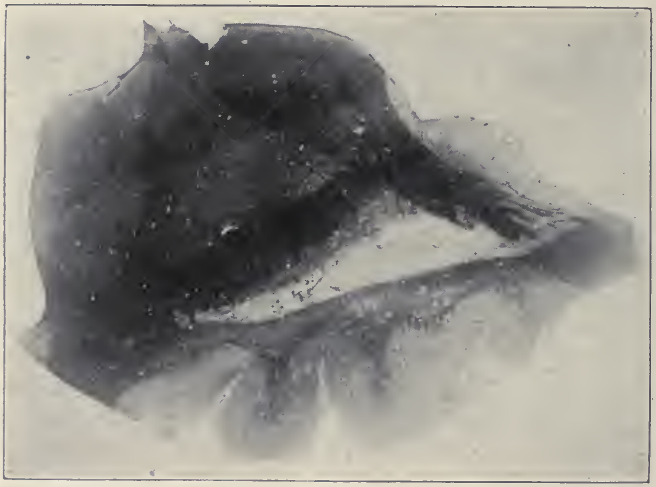

Fig. 4.—Basal ganglions from the case of double athetosis. Compare the lenticular nucleus with the lenticular nucleus in Figure 3. The section is from the left cerebral hemisphere.

In Pollock's[6] case of progressive lenticular degeneration, in addition to lesions of the lenticular nuclei some cortical changes were found but the Betz cells seem to have been little altered.

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4. Schütte: Arch. f. Psychiat. **51**:334, 1913.
5. Pfeiffer: J. Nerv. & Ment. Dis. **45**:289 (April) 1917.
6. Pollock: J. Nerv. & Ment. Dis. **46**:401 (Dec.) 1917.

Pseudosclerosis.—The pseudosclerosis of Westphal and Strümpell apparently is generally regarded by German writers as identical or at least closely allied with Wilson's progressive lenticular degeneration, and some go so far as to refer to both conditions collectively as the Westphal-Strümpell-Wilson syndrome (Thomalla). Wilson recognized the resemblance between the two disorders in an appendix to his paper in *Brain* and in Lewandowsky's "Handbuch," and Strümpell has expressed the opinion that they are the same disease.

Otto Maas[7] came to the conclusion that no distinction could be made clinically or pathologically between the two disorders. Thomalla[8] says that in all cases of pseudosclerosis carefully studied histologically, changes were found in the lenticular nucleus, in other ganglions of the cerebrum and often in the nucleus dentatus. He asserts that von Maas and von Dziembowski have demonstrated the identity of the two diseases. I have not been able to obtain the paper by the latter author, but Thomalla referring to it says that von Dziembowski in his first case found no changes in the lenticular nucleus and attributed all the symptoms to disease of the liver and alteration of the blood; nothing is said about the method of the histologic examination.

It is therefore worth while to study some of the recent papers on pseudosclerosis. This is a term used chiefly by German writers.

Hösslin and Alzheimer,[9] in a case of pseudosclerosis, found the convolutions in general small and the sulci widened. Nowhere was cellular infiltration found. The glia of the brain was altered everywhere and there were many large glia cells. The entire central nervous system was affected, especially the corpus striatum, thalamus, regio subthalamica, pons and nucleus dentatus. The cells after alcohol hardening were stained with toluidin blue.

A. Westphal[10] in his case of this disease found alteration of the glia nuclei, especially in the basal ganglions and the nucleus dentatus of the cerebellum, corresponding with those described by Alzheimer, but less pronounced and indicating an earlier stage of the disease.

Otto Mass' case of the same disease showed bilateral degeneration of the lenticular nucleus and nothing pathologic in the optic thalamus and caudate nucleus. In the lenticular nucleus the nerve cells had almost entirely disappeared, but nerve fibers coming from outside the lenticular nucleus and entering it were preserved. The neuroglia of the lenticular nucleus showed proliferation and many astrocytes.

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[7] Maas: Neurol. Centralbl., 1918, No. 1, p. 16.

[8] Thomalla: Ztschr. f. d. ges. Neurol. u. Psychiat. **41**:311, 1918.

[9] Hösslin and Alzheimer: Ztschr. f. d. ges. Neurol. u. Psychiat. **8**:183, 1912.

[10] Westphal, A.: Arch. f. Psychiat. **51**:1, 1913.

Maas says changes have been found in pseudosclerosis not only in the liver, but in the spleen and other glands. Fleischer, in reporting a case of pseudosclerosis, is said to have been the first to emphasize that liver changes belong to pseudosclerosis. Völsch later also pointed out the importance of the liver findings. Rausch and Schilder[11] say the liver changes in pseudosclerosis are identical with those in Wilson's progressive lenticular degeneration. The brain changes are more diffuse in pseudosclerosis, but Stöcker shows that this is not always true as he found changes in the entire brain in Wilson's type.

Rumpel[12] believed the liver which he studied from a case of pseudosclerosis was the result of fetal developmental disturbance, probably from congenital syphilis.

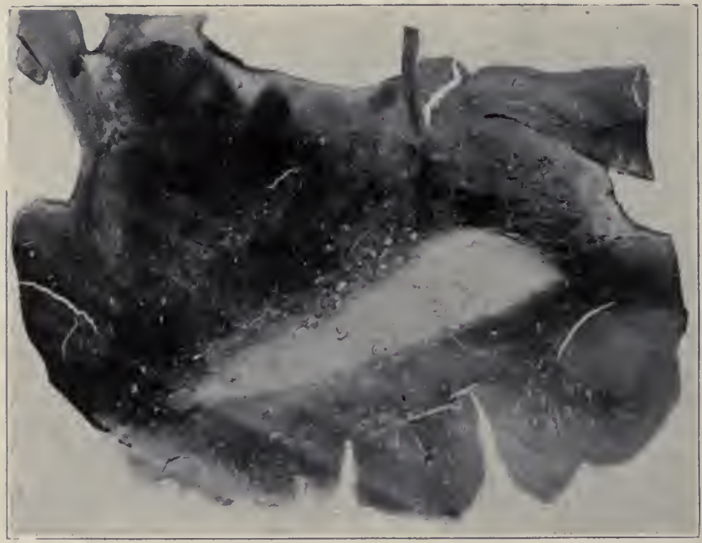

Fig. 5.—Section through the right basal ganglions from the case of double athetosis. The degeneration affects the putamen and the tissue consists of loose neuroglia. There is no large cavity in the putamen. It has been difficult to photograph these sections.

Athetosis and Lenticular Degeneration.— Are the changes of the nerve cells of the cerebral cortex independent of the alteration of the lenticular nucleus or are they secondary to it? It has been generally believed that no important connection exists between the ceberal cortex and the lenticular nucleus, and although this view has been difficult to accept, anatomic investigations have seemed to establish it. Wilson holds that the direct connection of the corpus

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11. Rausch and Schilder: Deutsch. Ztschr. f. Nervenh. **52**:414, 1914.
12. Rumpel: Deutsch. Ztschr. f. Nervenh. **49**:54, 1913.

striatum with the cerebral cortex is minimal. Recently von Economo,[13] however, has thrown much light on this question. He believes he has demonstrated an anatomic connection between the lenticular nucleus and certain parts of the cerebral cortex, a striotemporal and a strioparietal connection. This, he says, is contrary to the teaching of Déjerine, who denied connection from the cortex to the corpus striatum, and said that only collaterals from the pyramidal tract fibers entered the corpus striatum, as no cortical lesions caused atrophy of the corpus striatum, such as is seen in the optic thalamus. Von Economo holds that as there are association tracts between different parts of the cerebrum, and a lesion of one part does not cause atrophy of other parts connected with it by these association tracts, so there exists a connection between the corpus striatum and the cerebral cortex, and the tracts discovered by him, the striotemporal and strioparietal, are examples of such an association. It would be strange, he thinks, if the corpus striatum were isolated from the rest of the cerebrum except by its striofugal connections with the optic thalamus, corpus Luysii and nucleus ruber. Von Economo regards the tonic spasm without reflex exaggeration as a pathognomonic symptom of disease of the lenticular nucleus.

It is possible, therefore, that the alteration of the cells of the cerebral cortex has a close connection with the alteration of the lenticular nucleus. The changes in the Betz cells in my case of athetosis, evidently far greater than any alteration of the cortex observed by Wilson in his cases, may have some connection with the greater severity of involuntary movement in my case, in which there was athetosis of an extreme type in comparison with the tremor of Wilson's cases; and yet Madame Vogt did not find intense change in the cortical nerve cells in her studies.

The most striking difference in the description given by Wilson of progressive lenticular degeneration and one of acquired athetosis is in relation to the duration of the disorder and the presence of tremor, as in bilaterial athetosis the involuntary movement is not regular and is unlike that described by Wilson; and yet in Gowers' case which Wilson accepts as belonging to his type, it is stated that the fingers at times were extended and slowly moved in the irregular way characteristic of athetosis. Wilson states that tremor and athetosis may occur together, but they are sufficiently distinct to be described apart. In another place he distinctly states that associated movements and athetoid movements do not occur in progressive lenticular degeneration.

It remains to be proved, I think, whether the involuntary movements in progressive lenticular degeneration are practically always of the tremor type and are characteristic of lesions confined to the lenticular nucleus. My case of athetosis is proof to the contrary, so is Fischer's [14] case of athetosis and Thomalla's case of torsion spasm; especially so are those studied by Madame Vogt in association with others. Von Economo has shown by a case of his own and by others that in progressive lenticular degeneration spontaneous movements may be entirely absent. Lenticular lesions may therefore cause involuntary movements of different types.

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[13] Von Economo: Ztschr. f. d. ges. Neurol. u. Psychiat. **43**:173, 1918.

Wilson states, in comparing his cases with those of Madame Vogt,[15] that in his cases there were no associated movements or any type of athetosis. These differences he regards as important, for progressive lenticular degeneration is a disease *sui generis,* and is not to be considered as a variety of "athétose double." Madame Vogt believes that the syndrome of the corpus striatum is an "athétose double pure," but Wilson is more inclined to associate tremor than athetosis with the syndrome.

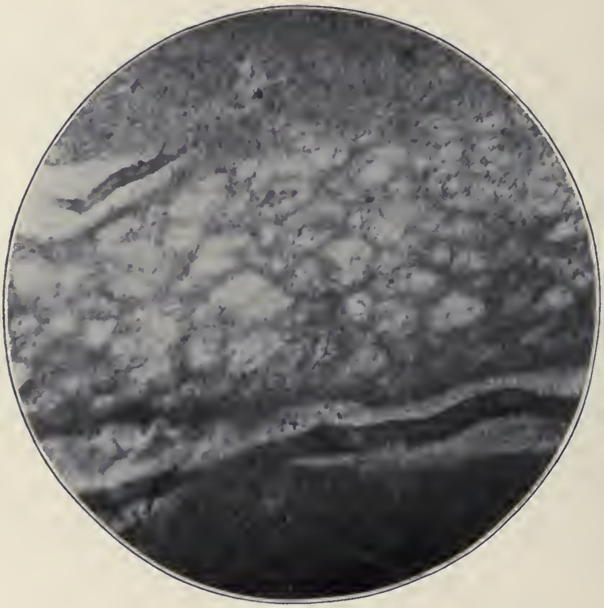

Fig. 6.—Section under greater magnification through the putamen, showing the loose neuroglia tissue. A blood vessel with an enlarged perivascular space is seen passing entirely across the section. (Photographs of the brain are by Dr. A. J. Smith.)

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[14] Fischer: Ztschr. f. d. ges. Neurol. u. Psychiat. **7**:463, 1911.

[15] Oppenheim and Vogt: J. f. Psychol. u. Neurol. **18**: 1912. Freund and Vogt: Ibid.

Wilson points out that in Anton's case, which he (Wilson) associates with cases of athetosis, the involuntary movements in the lower limbs were more like tremor. There was therefore in this case an association of tremor with lenticular disease.

Madame Vogt's findings are: In the case of infantile pseudobulbar paralysis with athetosis reported by her with Oppenheim, the caudate nucleus and putamen were much affected. The globus pallidus was atrophied in its dorsal portion. The ansa lenticularis appeared little altered. Nothing abnormal was found in the cerebellum or in the cerebral cortex. The internal capsule appeared to be unusually large on account of the change in the corpus striatum. Madame Vogt thought the parts destroyed must have had a regulating and inhibiting effect on the motor system.

In the case reported by her with Freund, the cerebral cortex showed no noteworthy alteration. Here also was found only atrophy and état marbré in a part of the caudate nucleus and the putamen. The rest of the brain was intact; even the Betz cells were normal.

Cécile Vogt, depending on the case of Anton, the cases she studied with Oppenheim and Freund and the two brains from Barré, believes the état marbré and atrophy of the corpus striatum produce the syndrome of the corpus striatum, namely, double athetosis without or almost without paralysis and without intellectual deficit or without pronounced intellectual deficit.

Strümpell[16] reports two clinical cases in brother and sister which he does not fully identify with Wilson's type, but brings into close relationship with it. Rigidity was the most characteristic feature in his cases and tremor was so slight and fine that without careful observation it would have been overlooked, and it was not always present. In one of these cases athetosis was especially marked in the hands and less so in the feet; in the other case athetosis was not observed in the hands, but it was pronounced and constant in the toes though not intense. These cases show the relation between progressive lenticular degeneration and bilateral athetosis.

Anton's[17] case was one of chorea with athetosis. He found almost symmetrical isolated lesions of the posterior half of the putamen of the lenticular nucleus. The optic thalamus and internal capsule were normal. He attributed the lesions of the putamen to thrombosis of the several small vessels supplying the putamen and passing through the substantia perforata. Such thrombosis might be caused in children by cachexia and fever. He quotes Kolisko as showing that the

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16. Strümpell: Deutsch. Ztschr. f. Nervenh. **54**:207, 1915-1916.
17. Anton: Jahrb. f. Psychiat. **14**:141, 1896.

arteries supplying the caudate nucleus and the globus pallidus come at a sharp angle from the anterior cerebral artery, and the current is opposite to that in this artery.

Oskar Fischer mentions that although lesions in a location typical for hemichorea are frequent, hemichorea seldom occurs. Why in one case chorea occurs and in another is wanting he cannot explain. The same kind of lesion in the same location in one case may cause hemichorea, in another hemiathetosis or hemitremor. There is no satisfactory explanation for this.

He reports a case of double athetosis of gradual development in which he found granular pigmentary degeneration of the ganglion cells in the globus pallidus of each lenticular nucleus. The boy was normal until 14 or 15 years of age, but between 15 and 17 the athetosis probably developed. The necropsy showed great diminution in the size of the globus pallidus, it appeared shrunken and was of brownish color, and was more intensely colored in its anterior portion, but showed no softening or hemorrhage. The atrophy of the lenticular nucleus was evident in Weigert sections. The cerebral hemispheres were smaller than normal, and the white matter less developed, but this was much less than the atrophy of the lenticular nucleus, although the atrophy was not equally intense in all parts of the nucleus. The putamen was distinctly atrophied but not nearly so much as the globus pallidus, which was about half the normal size. The globus pallidus was poor in medullated fibers, more so in the anterior portion, which was of much the same color as the putamen. The optic thalamus and internal capsule were distinctly smaller but the caudate nucleus showed no change.

The pallor of the globus pallidus was caused by a scarcity of medullated fibers and the small size of the medullated fibers preserved. The ganglion cells of this structure were greatly changed; in Nissl stain they were enlarged and rounded, and the nucleus was swollen and the nucleolus enlarged. The cell body contained numerous fine granules and other changes were apparent. The globus pallidus alone showed pathologic change. No changes were found in the anterior cerebellar peduncles.

LOCATION OF LESIONS IN ATHETOSIS

Schilder[18] shows that in athetosis the lesion was found in different locations, and he classifies the cases as follows:

Lenticular Lesions:

1. Anton's case in which pathologic changes were found only in the putamen of each lenticular nucleus and the left ansa lenticularis was small.

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[18] Schilder: Ztschr. f. d. ges. Neurol. u. Psychiat. **7**:219, 1911.

2. Berger's case in which athetosis was confined to the right side. A cavity was found in the left lenticular nucleus.

3. Oppenheim and Vogt's case in which there was pseudobulbar palsy with athetosis and changes in the corpus striatum.

Optic Thalamus Lesions:

1. Herz's case, in which there was athetosis on the right side. A cyst was found in the optic thalamus; the nucleus ruber, anterior cerebellar peduncle and nucleus dentatus were altered.

2. Muratow's case of athetosis. A gumma was found in the optic thalamus and the anterior cerebellar peduncle also showed a lesion.

Lesions were found in the nucleus ruber in the cases of Halban and Infeld, and Marie and Guillain.

The anterior cerebellar peduncle was affected in Bonhöffer's case.

A tubercle was found in the cerebellar hemisphere in Pineles' case.

In a case of unilateral athetosis reported by Schilder a tumor was found in the lenticular nucleus. Another lesion was found in the cerebellum.

Nonne has recently expressed the opinion there can no longer be any doubt that lesions of the optic thalamus and corpus striatum are responsible for athetosis and certain forms of chorea and paralysis agitans.

DYSTONIA MUSCULORUM PROGRESSIVA IN CORPUS STRIATUM GROUP

It is interesting to see that dystonia musculorum deformans has a pathology placing the disorder in the group of corpus striatum diseases.

In a case of this kind reported by Curt Thomalla there was intense alteration of the putamen of the lenticular nucleus. The putamen on each side was abnormally small; its tissue was below the level of the surrounding structures in a section through the brain; it was of softer consistency and was fissured (zerklüftet). The putamen showed softening, its parenchyma was completely destroyed, ganglion cells and nerve fibers had almost entirely disappeared, and the neuroglia had only partially proliferated to take its place. A spongy porous glia structure replaced the normal tissue of the putamen, in which the glia nuclei were much multiplied, and numerous fatty granular cells were in its meshes. Astrocytes were few. The vessels were conspicuous in this gliar reticulum, but were not quantitatively increased. A feeble round cell infiltration was found in the outer coat of certain vessels. The pathologic condition was sharply defined from the cortex of the island of Reil.

The nucleus caudatus showed no noteworthy change, and the bands of tissue connecting the nucleus caudatus and the putamen through the anterior limb of the internal capsule were not altered. The globus pallidus was not appreciably altered and the cortex of the island of Reil was intact.

The liver was in a condition like that described by Wilson in his cases and in cases of pseudosclerosis. The inner structure was different and was believed to indicate a defect of development. The pathologic findings were reported only in a preliminary manner.

Thomalla's patient showed transitory but typical athetoid movements, and he refers to the fact that double athetosis has often been mentioned in association with torsion spasm. His case of torsion spasm is the first in which there has been a necropsy examination. He says the alteration of the lenticular nucleus was almost identical with that in Wilson's cases, as was also the alteration of the liver.

He quotes Oulmont as saying double athetosis is a primary bilateral hemiathetosis, almost always beginning in the first years of life, usually with idiocy. Lewandowsky believed it to be more than bilateral hemiathetosis, but on account of deficient power of dissociation it is a form of generalized but not identical associated movements. There is a rhythmical character and slowness of the athetoid movements, spasmus mobilis, strongly influenced by emotion, involvement of the face, lack of dissociated movements in the face, diplegia, disturbance of reflexes and a positive Babinski reflex. Often there are contractures. The irritative motor phenomena may persist when the patient is quiet, even in sleep. Imbecility is almost constant, but many authors have observed that the intelligence is intact.

Thomalla speaks of the clinical resemblance of his case of torsion spasm to double athetosis and says Ziehen and Schwalbe also observed the similarity between their cases and athetosis. Oppenheim also accepted a relationship between these two disorders, and spoke of transitional cases. The relationship has been recognized also by Flatau, Sterling and others.

Thomalla points out that the Babinski reflex was absent in Klempner's case of double athetosis. He regards his case as a transitional one between torsion spasm and double athetosis, as he does also Flatau's and Fischer's cases. He says the liver in A. Westphal's case of pseudosclerosis was like the liver in his own case. He calls attention to the resemblance between torsion spasm and Wilson's progressive lenticular degeneration in the muscular rigidity of the entire body, including the muscles of mastication and speech; in the predominance of the flexors with gradually developing flexor contracture, etc. The resemblance between double athetosis and Wilson's type is shown by the muscular rigidity, the gradual development of contracture, the dysarthric and dysphagic disturbances, progressive character, family occurrence and psychic disturbances.

He discusses the resemblance between bilateral athetosis, pseudosclerosis, progressive lenticular degeneration and torsion spasm. In all there is disturbance of tonus shown by: spasms in double athetosis and torsion spasm; muscular rigidity in Wilson's type and pseudosclerosis, differing in all these forms from spasms of pyramidal tract origin; involuntary movements, disturbances of speech and swallowing in all, gradually developing contracture of the limbs in all, even to complete rigidity, masklike face and boardhard abdominal muscles. In all there is disease of the lenticular nucleus. The cases of double athetosis with pyramidal tract involvement must be separated from the group. Thomalla proposes for the whole group the name of dystonia lenticularis. He says as yet similar findings in the liver of patients with double athetosis do not exist; perhaps they have been overlooked; perhaps they are really lacking. In all the groups there is absence of atrophy, paresis, electrical alteration or pyramidal tract symptoms. In many cases cerebral cortical changes have been found.

J. Ramsay Hunt[20] found as the cause of juvenile and early adult paralysis agitans progressive atrophy of the globus pallidus, consisting of atrophy and diminution in number of the motor cells of the globus pallidus, the basal ganglion of Meynert, and especially the cells of similar type scattered through the caudate nucleus and the putamen (neostriatum). The evidences of cellular atrophy were especially well marked in the large cells of the neostriatum. There was a corresponding increase of the glia nuclei and a thinning of the fibers of the ansa system—striohypothalamic radiations. The small ganglion cells of the neostriatum were intact and the pyramidal tracts showed no signs of atrophy or degeneration. Hunt found no essential lesions except of the corpus striatum in juvenile paralysis agitans.

CAUSE OF INVOLUNTARY MOVEMENTS

Strümpell explains tremor and athetosis as the result of faulty innervation of the muscles which fix a limb in any position, when the agonistic and antagonistic muscles are not simultaneously innervated to the normal degree. If the innervation of one group occurs at a different time from that of the other, involuntary movement results; tremor if the alternate innervation is regular; athetosis if it is irregular. The proper innervation of the agonistic and antagonistic muscles of a part which permits proper fixation of the part is called by Strümpell myostasia, whereas the disturbance of this innervation so that steady fixation is impossible, is called the amyostatic syndrome or myastasia. Paralysis, ataxia, spasm, which are disturbances of muscle movement, he calls in distinction, the myodynamic complex or the pyramidal tract syndrome. In the type described by Wilson the amyostatic syndrome is especially marked, as shown by tremor, muscular rigidity, rigidity of expression, and other symptoms.

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[20] Hunt: Brain **40**:58, 1917.

Strümpell says that intestinal symptoms, dyspepsia, diarrhea and abdominal pain have been observed only in the pseudosclerosis or the type described by Wilson. This discussion by Strümpell is interesting, but giving all the types the name of amyostatic syndrome does not explain why they occur. This question has puzzled all students of the subject. Gowers attributed involuntary movements to lesions of the basal ganglions. Von Monakow does not believe tremor is caused by direct irritation of the pyramidal tract. He believes lesions of the corpus striatum cause irritation of the motor cortex and that from this source arise the impulses for choreiform movement. Wilson attributed involuntary movements to extrapyramidal fibers, such as are (1) in the tract from the nucleus dentatus through the superior cerebellar peduncle, to the nucleus ruber of the opposite side and from this to the outer part of the thalamus opticus and to the sensory and motor cortex. Involvement of this tract must cause athetoid and choreiform movements.

(2) In the tract arising in the nucleus lenticularis, through the ansa lenticularis in the regio subthalamica, the corpus Luysii and the nucleus ruber, from here as von Monakow's tractus rubrospinalis to the cells of the anterior horn of the spinal cord.

The movements are supposed to occur because of loss of corticopetal impulses which should pass to the cortex through the cerebello-rubro-thalamo-cortical tract. Lesion of this tract anywhere will cause posthemiplegic chorea by preventing the inhibitory impulses from reaching the cortex. This view is confirmed by the statement of deVries that extirpation of motor cortical areas in those with hemiparesis and choreo-athetoid movements causes cessation of these movements. Wilson ascribes the tremor to a destructive lesion of the lenticulo-rubro-spinal system. On account of the loss of this secondary motor tract the normal inhibiting impulse which the corpus striatum exercises over the anterior horn cells of the spinal cord are lost. The uniform innervation of the anterior horn cells is thus made impossible and stronger impulses pass to them by the pyramidal tract. Every voluntary movement increases the tremor, and this is usually true of the athetoid and choreiform movements.

HERNIATION OF THE BRAIN *

ADOLF MEYER, M.D.

BALTIMORE

The falx and tentorium constitute an important protection against any sudden impacts of pressure by keeping apart heavy portions of the brain, but they also provide an opportunity for trouble in case of swelling or need of displacement.

From the material that has gone through the Neurological Laboratory of the Henry Phipps Psychiatric Clinic, I have selected some cases illustrating various types of herniation under the falx and under the tentorium and into the foramen magnum. They give an interesting quasi-experimental picture of the topography and at least in two instances collateral consequences of possible importance among the so-called distant symptoms of brain tumor.

The line along which the tentorium can produce depressions passes over the uncus along the subiculum of the cornu ammonis.

The case A. M., with glioma of the left opercular region, shows, beside a moderate cerebellar wedging into the foramen magnum, a characteristic sagittal "tentorial line" on the left uncus, not to be taken for a sulcus semi-annularis Retzius (Figs. 1 and 2).

The case D. W., with a glioma of the anterior and middle hindbrain segment, shows similar tentorial lines due to slight hydrocephalus (Figs. 3 and 4).

A most extensive prolapsing glioma of the left temporal lobe (G. McL.) with death in coma, shows a frontal herniation under the falx and a marked subtentorial herniation of the uncus (Fig. 5).

In M. B. (Figs. 6 to 8) a cancer metastasis with a cyst in front of the paracentral lobule leads to herniation of the fornicate gyrus under the falx and subtentorial herniation of part of the uncus and of both subicula, with a striking impingement on the calcarine branch of the posterior cerebral artery and collapse of the corresponding part of the occipitotemporal cortex; hence providing a foundation for hemianopsia.

In R. H. (Figs. 9 and 10), an aneurysm of the circle of Willis just in front of the optic tract and chiasma impinged on the circulation of the right hemisphere, with swelling of the right hemisphere without actual softening or infarction, but marked herniation of the subiculum; left hemiplegia and hemianesthesia, three days later coma, and nine days later death.

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* Read at the Forty-Sixth Annual Meeting of the American Neurological Association, New York, June, 1920.

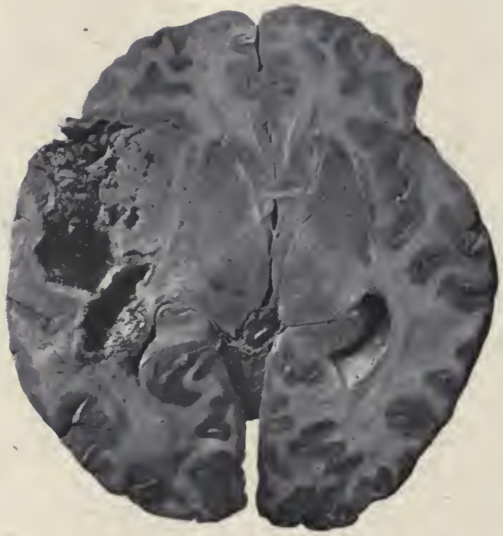

Fig. 1.—A. M., glioma with cystic degeneration in the left opercular region.

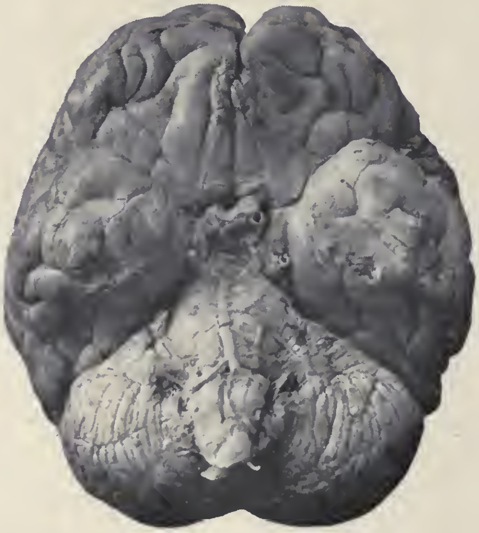

Fig. 2.—A sagittal depression of the uncus on either side marking the line of the tentorium; also moderate wedging of the cerebellum into the foramen magnum.

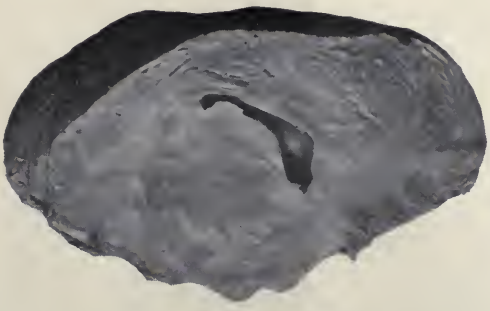

Fig. 3.—D. W., general extent of the glioma bulging into the hind-brain ventricle.

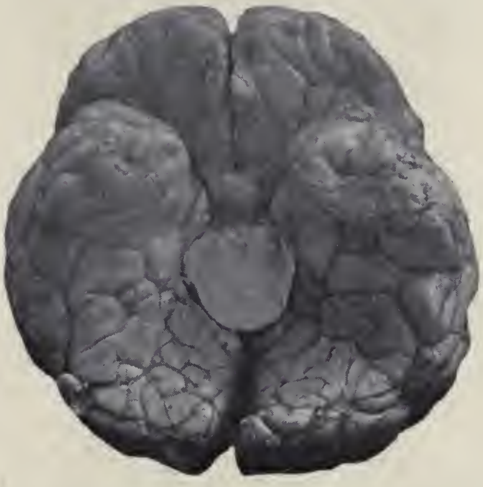

Fig. 4.—D. W., hydrocephalus visible in the distention of the tuber cinereum. Change of contour of the isthmus through the glioma; marked sagittal depressions of each uncus.

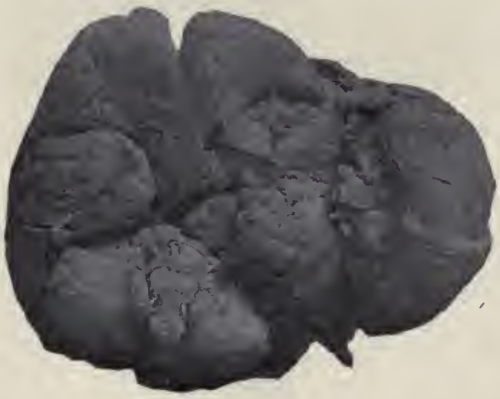

Fig. 5.—Prolapsing glioma (G. McL.); herniation of the left frontal lobe under the falx. Almost the entire left uncus is displaced on the inner side of the tentorium.

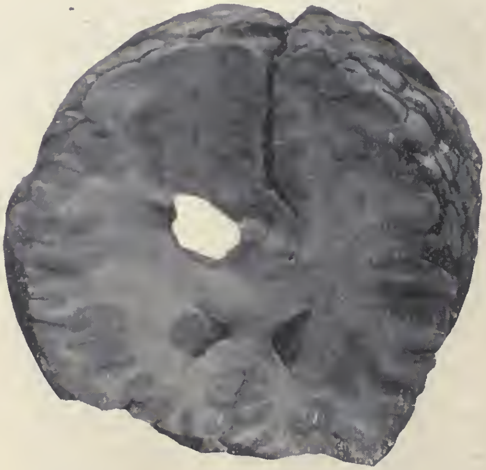

Fig. 6.—M. B., metastatic cancer and cyst taking up the mesial edge of the hemisphere. The lateral ventricles are somewhat compressed. The fornicate gyrus is pushed under the falx beyond the median line.

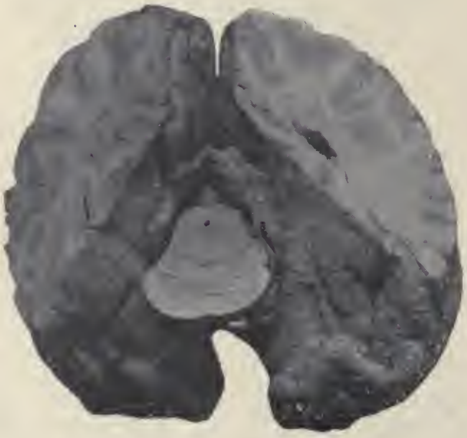

Fig. 7.—A ridge of herniation of the subiculum of the cornu ammonis to either side of the pineal region; compression, on the right side, of the posterior cerebral artery to the calcarine region; complete reduction of the calcarine and fusiform gyrus cortex on the right side, in contrast to the normally wide cortex on the left.

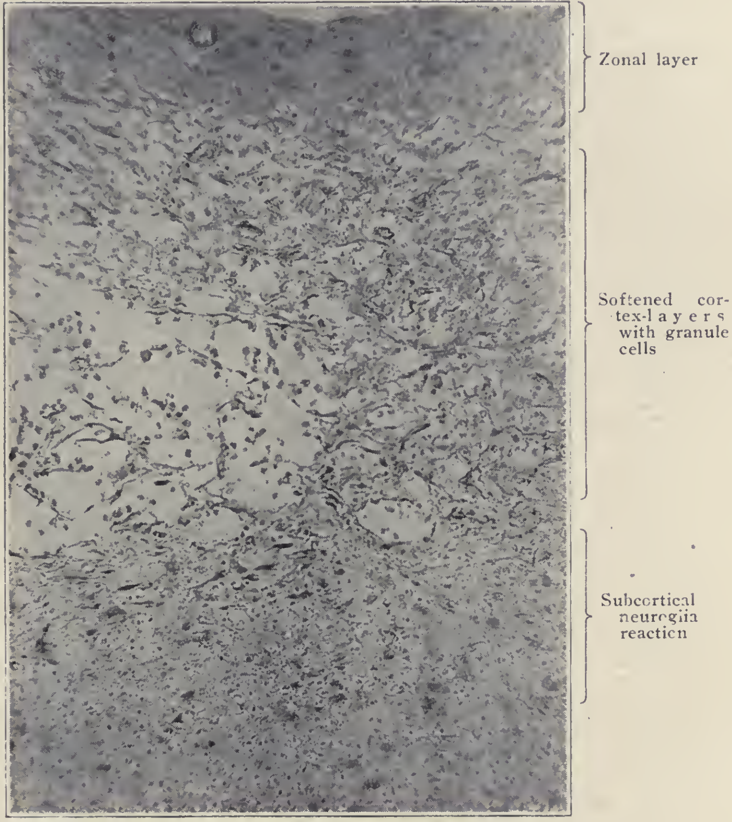
Zonal layer
Softened cortex-layers with granule cells
Subcortical neuroglia reaction

Fig. 8.—Calcarine cortex of Figure 7 with complete collapse of the cortex layers but preservation of the subcortex.

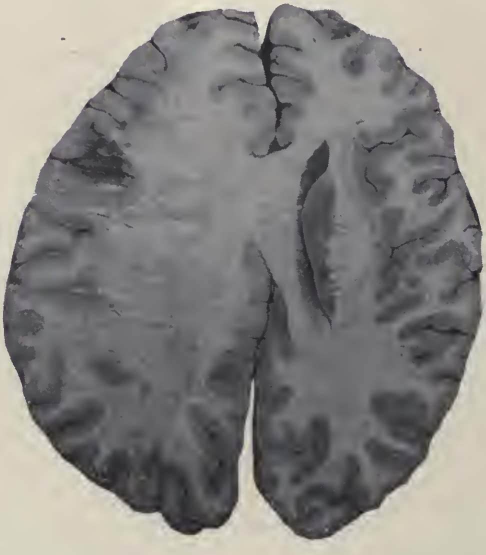

Fig. 9.—Swelling of the right hemisphere (to the left in the illustration).

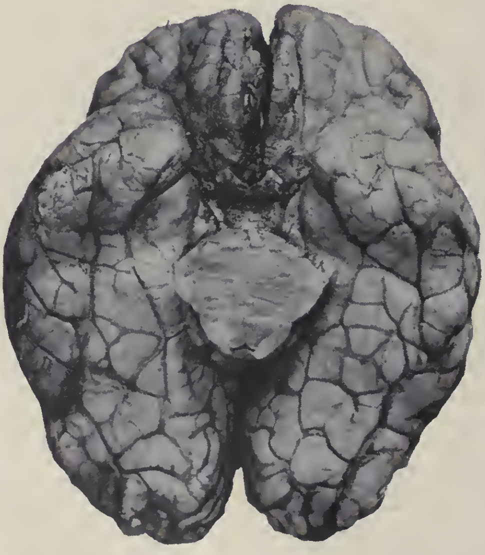

Fig. 10.—Hemorrhage from aneurysm in front of the optic chiasma; interference with the circulation in the right Sylvian artery; tentorial lines of the uncus and marked herniation of the subiculum (to the left of the midbrain).

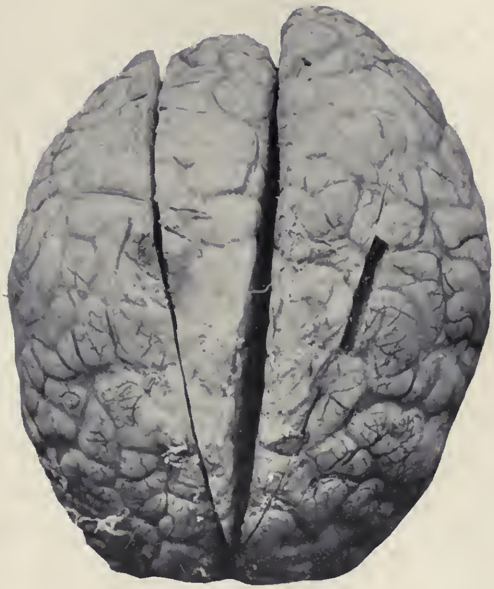

Fig. 11.—M. W., cancer metastasis in the right postcentral gyrus (to the left in the illustration).

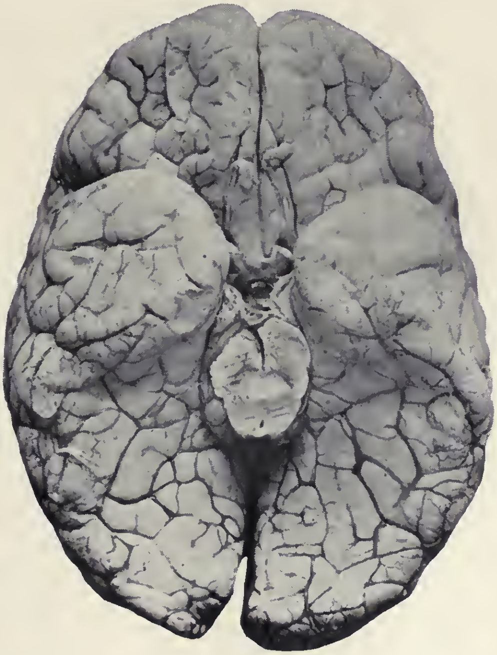

Fig. 12.—M. W., tentorial lines of the uncus and plain herniation of the subiculum along crus and midbrain.

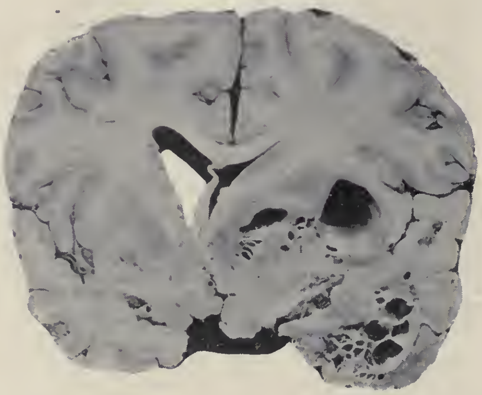

Fig. 13.—W. H. J., character of the cystic glioma extending from the striatum and frontal region through the temporal lobe to the ventral lip of the calcarine cortex (Fig. 16).

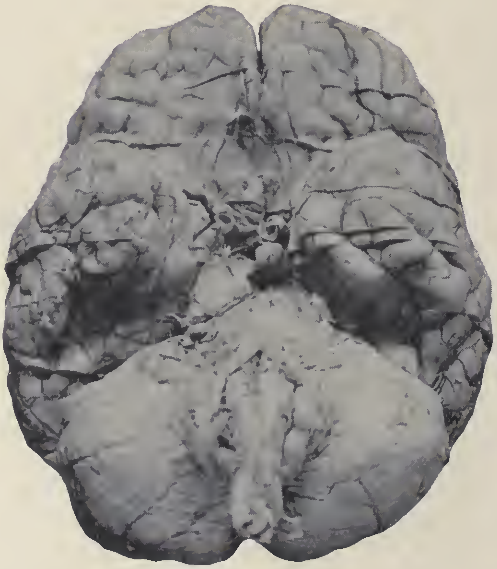

Fig. 14.—W. H. J., subtentorial herniation of the right uncus (to the left in the illustration) similar to that seen in Figure 5.

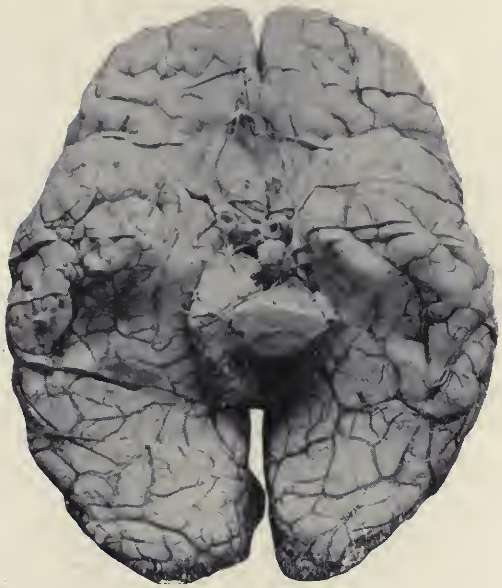

Fig. 15.—Continuation of the uncus hernia into a thick protrusion the entire length of the subiculum without occluding the posterior cerebral artery.

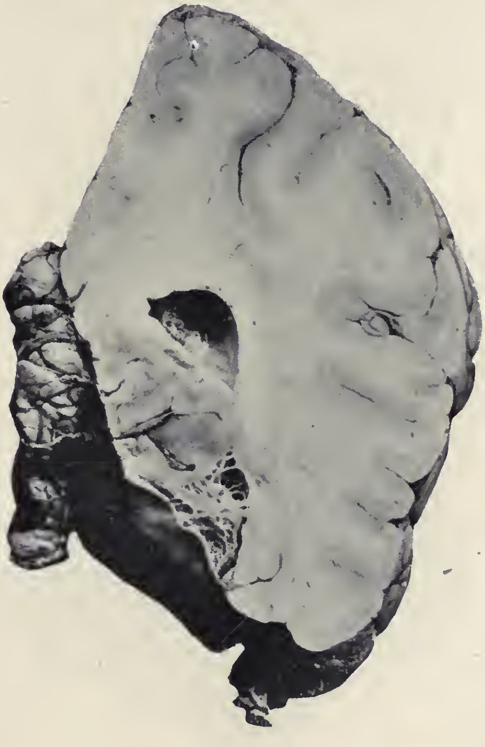

Fig. 16.—W. H. J., occipital horn and calcar avis. Cystic glioma of the basal lip of the calcarine. Prominent ridge of subiculum region forming a subtentorial herniation.

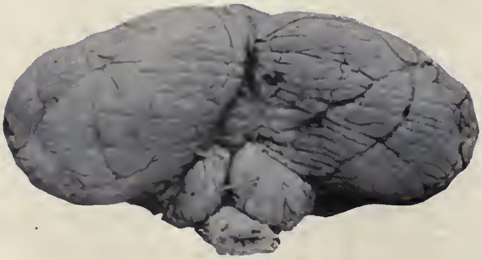

Fig. 17.—W. H. J., herniation into the foramen magnum.

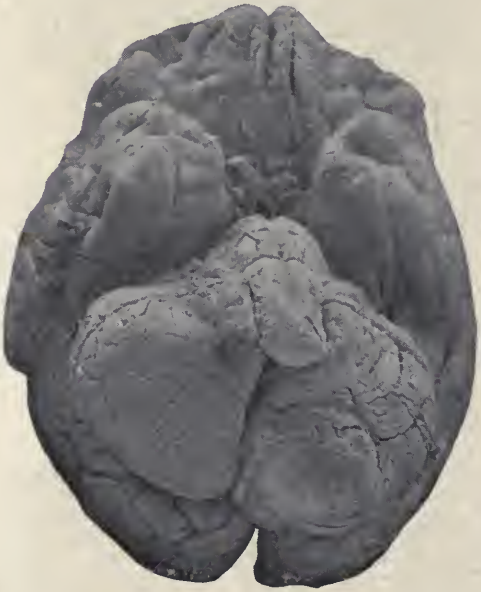

Fig. 18.—B. H., cancer metastasis of the left cerebellar hemisphere.

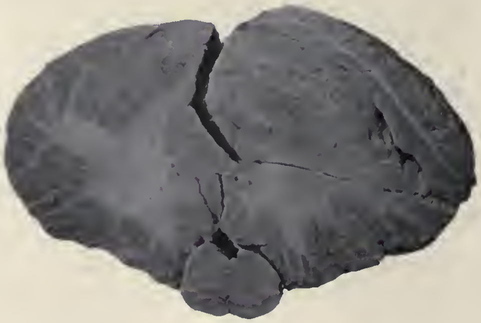

Fig. 19.—B. H., extent of the cancer.

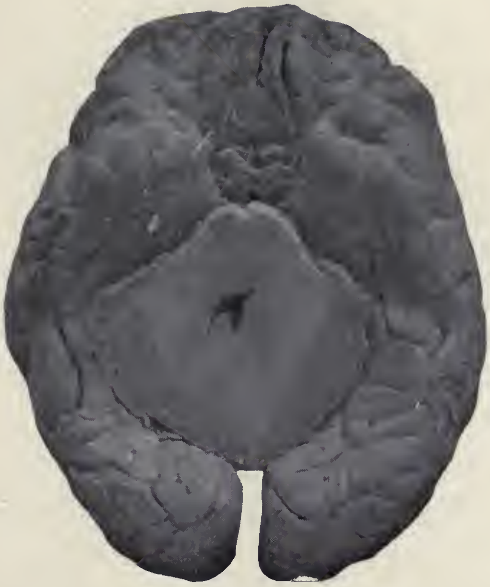

Fig. 20.—B. H., distortion of hindbrain ventricle by the cerebellar cancer.

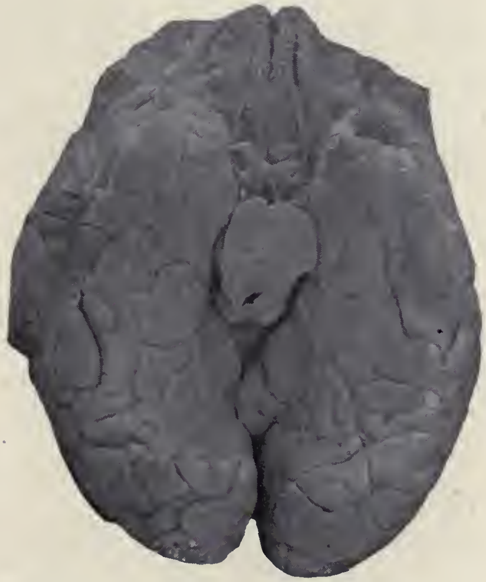

Fig. 21.—B. H., infratentorial distortion of the midbrain; remarkable supratentorial distention of the splenium and dislocation of the limbic lobe.

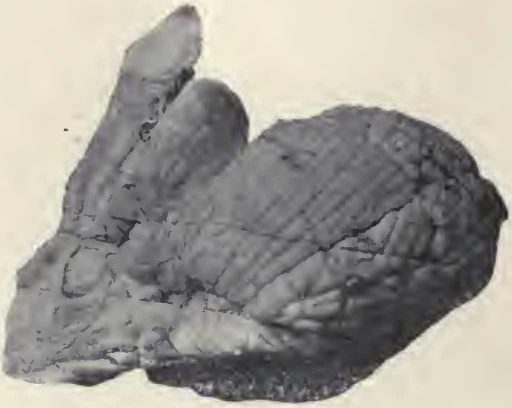

Fig. 22.—Foraminal wedging in case B. H.

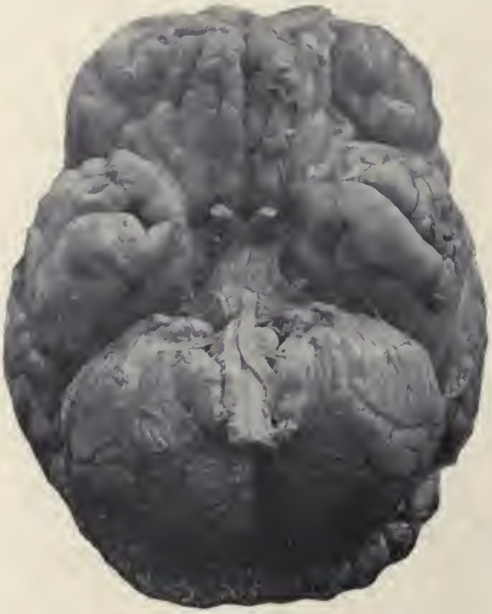

Fig. 23.—Child of 10 months; convulsions. Moderate cerebellar wedging in brain swelling.

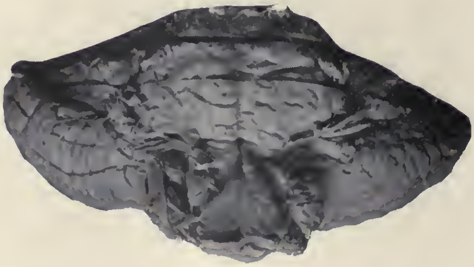

Fig. 24.—Marked wedging of the foramen, connected with lumbar puncture.

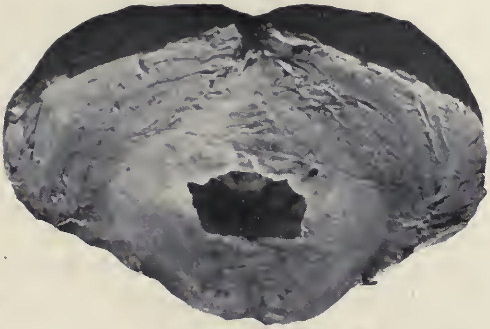

Fig. 25.—Distention of hindbrain ventricle in foraminal wedging.

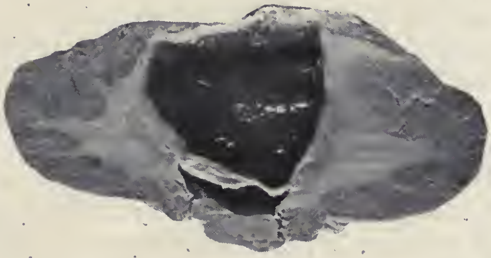

Fig. 26.—Cerebellar cyst causing foraminal herniation and partial compression of vertebral artery with bruit.

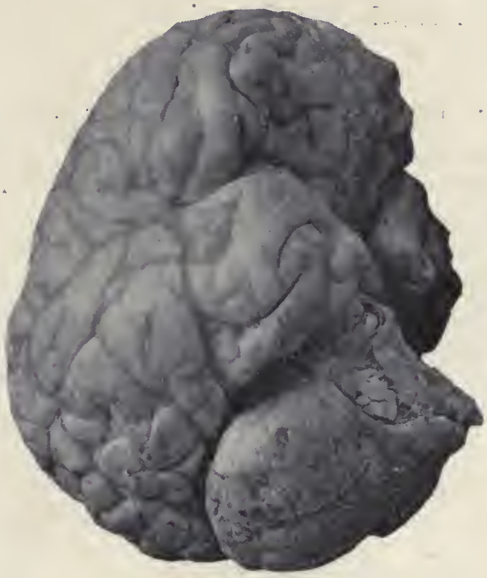

Fig. 27.—Herniation of cerebellum leading to compression of the vertebral arteries.

In M. W. (Figs. 11 and 12), an hemorrhagic cancer metastasis in the right postcentral gyrus led to subtentorial herniation and to wedging of the cerebellum.

An unusually great subtentorial herniation occurred in W. H. J. (Figs. 13 to 17), with a cystic glioma extending from the right frontal lobe to the right occipital lobe, without, however, occluding the calcarine artery. Wedging of the cerebellum was added as a secondary consequence.

In B. H. (Figs. 18 to 22), a cancer metastasis in the inferior semilunar lobule of the cerebellar hemisphere led to cerebellar herniation into the foramen magnum (Fig. 22), and to an interesting supratentorial distention of the region of the splenium, with death eight days after a subtemporal decompression.

Moderate wedging of the cerebellum (Fig. 23) in a child of 10 months, with convulsions and brain-swelling, but no focal lesion, led to a case of marked wedging of the cerebellum, distention of the hindbrain and forebrain ventricle (Figs. 24 and 25); severe frontal headache, lumbar puncture, followed by aggravation and disturbed vision and death in three weeks.

A counterpart to the case with secondary hemianopsia is produced by a case of my associate, Dr. Charles Bagley. The patient presented frontal headaches, convulsions, occasional staggering, but a vascular bruit over the entire cranium. A cerebellar cyst (Fig. 26) had produced a foraminal wedge which carried the vertebral arteries about 5 cm. below the foramen (Fig. 27).

CONCLUSION

Herniation in brain tumor and brain swelling occurs under the falx, under the tentorium and into the foramen magnum.

In the presence of pressure symptoms, we must remember that hemianopsia or vascular bruit may be a symptom of herniation.

A suspicion that herniations of the subiculum might be responsible for the sclerosis of the cornu ammonis in epilepsy through the effects of transitory subtentorial herniation is not, so far, corroborated by the present series.

CONTRIBUTION TO THE HISTOPATHOLOGY OF "PSEUDOTUMOR CEREBRI"

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INTRODUCTION

In 1904, Nonne[1] reported a series of cases which he tried to combine into a clinical entity. This was characterized by a more or less chronic development of the picture of brain tumor, with or without localizing signs, and without discoverable etiology. The patients usually recovered, either spontaneously or after treatment with mercury and iodids. If death occurred, no adequate macroscopic or microscopic lesions could be found. Nonne went so far as to claim that in some cases no microscopic pathology was present.

Nonne's publication was followed by numerous reports of similar cases in some of which the symptom-complex in question was referred to as "die Nonnesche Krankheit"[2] or "das neue Kranksheitsbild."[3] Prior to Nonne, a number of similar cases were reported by Quincke[4] under the name of serous meningitis.

It was well known that various pathologic conditions could simulate brain tumor, but the idea that at necropsy no pathologic changes or an unknown pathologic lesion might be found met with widespread skepticism. At the present time, such an opinion is practically abandoned, although Nonne in his last word on the subject[5] still maintains that such cases exist.

As foundation for this opinion, there could be found in the literature only two cases: Case 11 in Nonne's[1] series, and case 9 in Reichardt's.[6] Of these, Reichardt's bears clinically no resemblance to brain tumor,

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[1] Nonne: Ueber Fälle vom Symptomenkomplex von Tumor cerebri mit Ausgang in Heilung, Deutsche Zeitschr. f. Nervenheilk. **27**:169, 1904.

[2] Oppenheim and Borchardt: Zur Meningitis serosa chronica circumscripta (cystica) des Gehirns, Deutsche med. Wochschr. **36**:57, 1910.

[3] Finkelnburg: Ueber Pseudotumor cerebri, Zentralbl. f. d. Grenzgebiete d. Med. u. Chir. **15**:515, 1912.

[4] Quincke: Ueber Meningitis serosa, Samml. klin. Vortr. **67**:655, 1893 (Innere Med., no. 23).

[5] Nonne: Der Pseudotumor cerebri, Neue Deutsche Chirurgie **10**:107, 1914 (Die allgemeine Chirurgie der Gehirnkrankheiten, II Teil).

[6] Reichardt: Zur Entstehung des Hirndrucks und anderer Hirnkrankheiten (u. s. w.), Deutsche Ztschr. f. Nervenheilk. **28**:306, 1909.

but was a straightforward case of catatonia. In Nonne's case, one cannot help feeling that if the microscopic examination, which was very inadequate, had been more thorough, some changes would have been found. Cases studied microscopically with care have always shown pathologic changes. Only six such examinations have been recorded and they will be briefly reviewed.

REVIEW OF CASES WITH HISTOPATHOLOGIC REPORTS

Case 1.[7]—A man, aged 20, gave a history of gradually developing frontal headache, with defective hearing on the right, defective vision, double vision and lethargy.

Findings.—Atrophy of the optic nerves was found, the sense of smell was absent, the right corneal and conjunctival reflexes were sluggish, there were hyperesthesia of the upper branch of the right trigeminal nerve, and tenderness to pressure in the right suboccipital region. Diagnosis: tumor of the posterior fossa.

Necropsy.—No macroscopic pathologic change was discovered. Microscopically, widespread leptomeningitis (not tuberculous or syphilitic) over the base of the brain, and perineuritis as well as interstitial neuritis of the optic, olfactory and right acoustic nerves were noted. There were no changes in the brain except slight perivascular infiltration in the pons and medulla. Some polymorphonuclear leukocytes were seen around the eighth nerve.

Finkelnburg[7] intimates that this is the first time a case simulating brain tumor has been properly examined. The findings we accept, but certainly the clinical picture was stretched to make it a brain tumor.

Cases 2 and 3.—In these cases, studied pathologically by Rosenthal,[8] it is impossible to judge just how closely a tumor picture was simulated since the history and clinical examination are very incomplete.

Findings.—Stiffness of the neck, stupor, falling to the right, cerebellar gait, and optic neuritis were present in Case 2. Regarding Case 3, it is stated only that the disorder was of cerebellar nature with optic atrophy.

Necropsy.—Nissl's acute cell disease involved the cortex; there was an increase of lipoids in the ganglion cells; the glia cells were not much changed, those along the vessels contained much fat, and the blood vessels were much dilated; macrophages (Abräumzellen) with blood pigment were found; preameboid and ameboid glia cells were seen near the ventricles; there was an increase of subependymal glia fibers; the leptomeninges were slightly infiltrated with lymphocytes, blood pigment and greenish products of cell catabolism (Abbaustoffe).

The findings are those of lowgrade meningo-encephalitis with hemorrhages and reactive phenomena in the glia.

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[7] Finkelnburg and Eschbaum: Zur Kenntnis des sog. Pseudotumor cerebri mit anatomischen Befund, Deutsche Ztschr. f. Nervenheilk. **38**:35, 1910.

[8] Rosenthal: Histologische Befunde beim sog. Pseudotumor cerebri, Ztschr. f. d. gesamte Neurol. u. Psychiat. **7**:163, 1911.

Case 4.—A 14 year old boy gave a history of gradually developing left-sided weakness, psychomotor dullness, occasional headaches and vomiting.

Findings.—Left-sided spastic hemiparesis, uncertain gait, and increasing stupor were noted.

Necropsy.—There were no macroscopic changes. Microscopically, there were found marked lymphocyte and plasma cell infiltration of the adventitial spaces, with hyperemia and occasional hemorrhages; numerous large nucleated epithelial cells; occasional fat granule bodies (Körnchenzellen) and rare giant cells, especially in Langhans' sense. These changes were found around the small blood vessels of the right internal capsule, the neighboring lenticular nucleus, the dorsolateral part of the pes pedunculi, both subthalamic regions, the white substance of both cerebellar hemispheres and the nucleus dentatus.

The clinical picture in this case is again not quite convincing. Choked disk is absent and there were no epileptic fits. Yamakawa,[9] who recorded the foregoing case, came to the conclusion that the changes found belong to the category of atypical tuberculous connective tissue changes.

Nonne[5] reported two further cases with histologic examination, in the first by Spielmeyer and the second by Ranke.

Case 5.—A woman, aged 47, after a psychic trauma, had transitory attacks of vomiting and diarrhea, with pain in the left foot. Soon afterwards she had double vision and spots before the eyes. For eight months there was gradually lessening acuity of vision.

Findings.—The patient was able to distinguish only light and dark; the skull was somewhat tender to pressure over the forehead; bilateral high grade choked disk was observed.

Course.—Right decompression was performed, with resulting hernia cerebri and death.

Necropsy.—Spielmeyer found macroscopically greatly dilated ventricles. Microscopic examination revealed moderately widespread affection of the cortex with involvement of the white substance, without dependence of the affection on the blood vessels. In the cortex the fibers were badly damaged and often entirely absent over wide areas; the nerve cells showed many chronic changes, and a great increase of the glia cells (Trabantzellen). In the deeper layers of the cortex the glia fibers were greatly increased, but particularly in the white matter. The glia cells did not show the characteristics of glioma cells. They resembled those seen in secondary glia proliferation. The process involved especially the vicinity of the central fissure and the adjoining frontal region.

Spielmeyer stated (according to Nonne) that he knew of no sickness which could cause the changes found. It seems that they may be well explained by the operation, which resulted in a hernia of the brain, hemiplegia and other permanent organic lesions.

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[9] Yamakawa: Eine chronische disseminierte Encephalitis unter dem Bilde des sog. Pseudotumor cerebri, Mitt. a. d. med. Fakult. d. k. Univ. zu Tokyo **10**:201, 1913.

Case 6.—For four weeks a 35 year old man had occasional spells of vomiting, and for some time headaches which grew worse at night. Later there were double vision and spots before the eyes.

Findings.—Definite but not high grade choked disk with hemorrhages was observed, the pupillary reactions to light were sluggish and slight, the blood Wassermann reaction was positive, the pressure of spinal fluid was high, with a cell count of 25.

Necropsy.—Marked hydrocephalus internus and ependymitis granularis were present. Microscopic examination of the brain revealed cadaver changes (27 hours postmortem); infiltration of the pia with lymphocytes and an occasional mast cell; slight perivascular infiltration with lymphocytes and an occasional mast cell in the cortex, and very marked similar infiltration in the cerebellum, brain stem and basal ganglions; products of tissue catabolism (Abbauprodukte) in all regions, especially near the ventricles, where glia cells were present with homogeneous bodies containing lipoid material; widespread ependymal proliferation in all the ventricles; occasional fresh hemorrhages in the cortex, and proliferation of the intima cells without signs of fresh proliferation in many of of the blood vessels.

Ranke concluded that there was present a chronic inflammatory process, most probably syphilitic, with secondary hydrocephalus, and that the glia, ependymal and "Abbau" changes near the ventricles were probably secondary to the pressure of the ventricular fluid. From the clinical history one would rather have suspected that cerebral syphilis and not a tumor would have been found.

It will be noted from the foregoing reports, what would be even more evident if the histories of other cases not reviewed should be recorded, that full-fledged tumor pictures are rarely present. The localizing symptoms of tumor are usually very meager; choked disk is not very pronounced and often absent entirely.

REPORT OF AUTHOR'S CASE

The patient whose report follows presented a definite left cerebellar hemisphere syndrome and bilateral choked disk reaching from 3.5 to 4 diopters.

L. McN., a school girl, aged 16, was admitted to the Peter Bent Brigham Hospital (Surg. No. 11187), Sept. 19, 1919, complaining of vertigo, attacks of headaches and vomiting, poor vision, cross-eyes, weakness of left side, and instability in walking.

History.—The family history was negative. The patient's birth was normal. She was a little late in walking and her mother says that her head has always been rather large. She had diphtheria eight years ago and a mild attack of influenza last winter, but has never otherwise been seriously ill. She had no injuries and no operations. Catamenia was normal. Her habits were good.

Present Illness.—According to the patient's father, this began after an attack of influenza, November, 1918. While playing in church the notes became dim and she had a severe frontal headache which afterwards spread over the entire head, and she took to bed. The next day the headache was gone, but since then every week or so she had severe frontal headache afterwards passing over the entire head and lasting for a day or two. Later, these headaches localized in the back of the head, and the neck became sore at times. Her father noticed that when she had severe headache she staggered in walking and was apt to go to the left, and that she could not use her left arm and leg so well as the right. The patient, however, says that she had these headaches for several months before the attack of influenza, though never so severe. The father noticed that her left eye was turned in last March, but the patient's aunt said that the eye had been crossed for some time previously; just how long is not certain. About two weeks before admission, after an interval of freedom from headaches for three weeks, lumbar puncture was performed. This caused very severe headache which has never been entirely relieved and was made worse by assuming the erect posture, so that the patient has not been out of bed since. She was not so alert as formerly and at the time of examination was very stupid. Dr. Barrett of the Charles Sanitarium made a Wassermann test of the blood and spinal fluid, which gave negative results.

Physical Examination.—The patient was well developed and well nourished. Her brow was furrowed, she moaned from time to time as though in great pain, and also yawned occasionally, and rubbed her nose. The head seemed rather large and square, but there was no cracked-pot sound. There was suboccipital tenderness on both sides, perhaps greater on the left. No dilatation of the extracranial veins or exophthalmos was present. Neck, heart and abdomen exploration proved negative. The blood pressure was 108 systolic and 58 diastolic. The skin and temperature were normal.

Cranial Nerves.—Olfactory: Odors were recognized, and there were no subjective odors.

Optic: Fundus examination revealed on both sides that the disk margins were obscure, the disk being elevated about 1 diopter. The physiologic cup was filled, the veins were slightly engorged, and there was slight perivascular streaking, but no hemorrhages. There was bilateral choked disk, but no hemianopsia to rough test.

Motor Oculi, Patheticus and Abducens: There was downward and outward squint of the right eye with ptosis of the right eyelid. The pupils were equal and regular and reacted to light but not to accommodation. Movements of the right eyeball were restricted inward and upward. There was no nystagmus.

Trigeminal: No involvement of this nerve was manifested.

Facial: This nerve was normal.

Acoustic: No positive findings of disturbance of this nerve were elicited.

Vestibular: There was no dizziness; the Romberg sign and gait were not tried.

Glossopharyngeal: There was some difficulty in swallowing, particularly during the preceding days, and the voice had changed in character. The aunt thought it was harsher than usual.

Vagus: The pulse was regular but slow. The palate was symmetrical. There had been considerable vomiting.

Spinal Accessory: The trapezius was of normal power on the right side, somewhat weak on the left.

Hypoglossal: There was no deviation of the tongue and no tremor.

Cerebrum.—Frontal: The patient had lately become very stupid and said herself that she did not take as much interest in things as formerly. At the time of examination she could scarcely be aroused. Precentral: There were no convulsions or twitchings. Postcentral: No disturbance of touch, pain or temperature sensitivity, muscle sense or sense of position was noted. Temporal: There were no hallucinations of smell or taste, no uncinate attacks, and no hemianopsia. Occipital: There were no hallucinations of light and no hemianopsia. Parietal: Astereognosis was not present.

Cerebellum.—There was suboccipital tenderness on both sides, possibly slightly greater on the left. There was some stiffness of the neck and flexion caused some pain. There was no nystagmus. The Romberg test and the gait were not tried, but there was a history of staggering to the left and inability to stand with the eyes closed, the patient falling to the left. There was marked hypotonicity on the left side, more marked in the upper extremity. Holmes' wrist sign was positive, as was Holmes' finger sign also. There was marked adiadokocinesis in the left hand. Muscle sense and sense of position seemed to be fairly normal. There was marked ataxia of the left upper and lower extremities on finger to finger, finger to nose and heel to shin tests. Caloric and rotation tests were not tried.

Reflexes.—The biceps, triceps and periosteal reflexes, knee jerks and ankle jerks were elicited, being greater on the left than on the right. The corneal, epigastric, abdominal, and plantar reflexes were present, but the left plantar was very sluggish.

Vasomotor: No vasomotor disturbance was observed.

Sphincters.—The control of these was good.

Roentgen-Ray Examination.—The convolutional impressions were increased throughout the skull. The sella was not well outlined. The frontal bone appeared irregularly thin, the parietal bones were thickened and the skull in this region was irregular in outline.

Operation.—Suboccipital exploration for presumed cerebellar tumor was performed by Dr. Harvey Cushing, Sept. 23, 1919, the anesthetic being ether.

The usual cross-bow incision was made. The suboccipital bone was much thinner on the left, though there were no patches of exceptional thinning. A wide area of bone was removed. There was an exceptionally deep bony septum projecting between the two hemispheres for a depth of about 1 cm. Some difficulty was experienced in dislodging this median partition and in doing so the dura near the lower part of the field was slightly torn and cerebrospinal fluid escaped abundantly through the small opening. There being no further risk in incising the dura, this procedure disclosed two normal appearing hemispheres. It is possible that there may have been some wedging of the cerebellum into the foramen, though this was not extreme, and on pulling up the two hemispheres the calamus could be seen. The right hemisphere was larger and possibly more firm as though there might have been a tumor. An exploratory needle was inserted into each hemisphere to a depth of 5 cm. without disclosing a lesion (possible cyst). The patient took the anesthetic well, and the wound was quite dry throughout. It was closed in layers, but just before the final closure a perforation was made through the pole of the left hemisphere and the needle was inserted into the ventricle, an abundance of fluid being reached at a depth of 3 cm., evidencing therefore an internal hydrocephalus.

Postoperative Course.—The patient made a good ether recovery. Five days after the operation she was much brighter; the headaches had been entirely relieved, but the ptosis of the right eyelid persisted.

The cast was removed October 3 and the stitches taken out. The wound healed in excellent approximation, but some fluid was under the incision line.

Headaches and vomiting were completely relieved, and the patient thought that her vision was better, but there was no improvement in ability to use the left arm and leg.

The patient was up in a wheel chair October 5. Examination of the left fundus disclosed that the disk and the vessels over the disk were obscured by new tissue, the veins were tortuous, the disk was not elevated. The same conditions were noted in the right fundus, with small hemorrhage in the upper border of disk.

The patient was very well and happy until the morning of October 10. While sitting up in a wheel chair she became dizzy and faint, was put back to bed and recovered. After lunch she became faint and stupid and vomited. Lumbar puncture showed that the fluid was not under tension, and perfectly clear. About 6:30, the patient had a generalized convulsion. There were clonic movements of all the limbs beginning on the left side, but she did not lose bladder or rectal control. The respiration and heart rates continued normal.

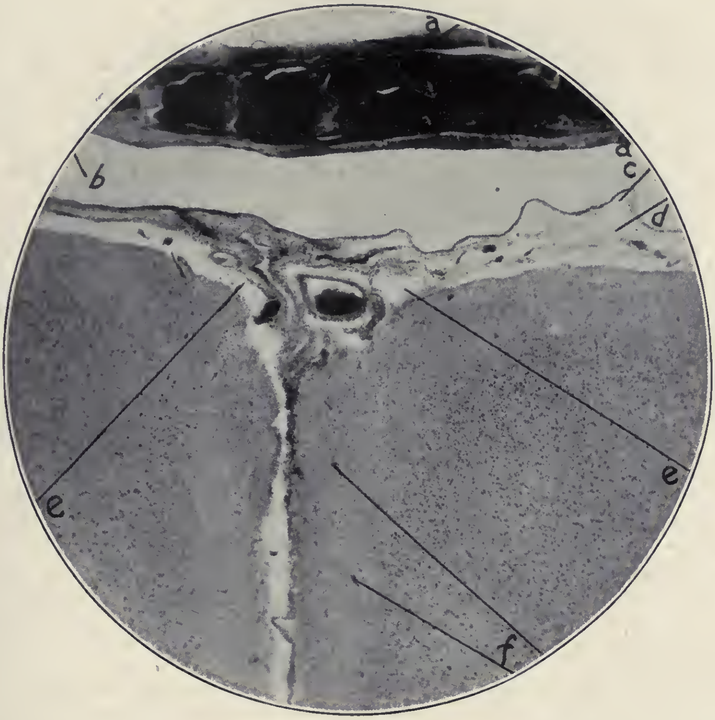

Fig. 1.—Transverse section from the motor area near the longitudinal sinus, the three membranes included. *A,* inner and outer layers of the dura, the cartilaginous mass between being very darkly stained and connective tissue strands dividing it being distinctly seen. *B,* subdural space. *C,* arachnoid membrane. *D,* subarachnoid space, greatly infiltrated, especially in the region of the sulci. *E,* so-called epicerebral space (probably an artefact, though some amorphous substance is seen within the space said to be the result of edema). *F,* small vessels. (Celloidin section, thionin stain, ×24.)

On the following day the patient had another convulsive attack about 3 a. m., followed by profuse perspiration. She was fairly bright and said her headache was better. October 16, she was very dull all day and could not be induced to talk.

October 19, there was bilateral ptosis and bilateral downward and outward squint with paralysis of convergence, both more marked on the right. The right pupil was slightly larger than the left. On the left side the reflexes were slightly exaggerated with marked hypotonicity and ataxia. The decompressed area was full, but not especially tight. No sensory disturbance was elicited. The Babinski reflex was negative. Both disks were very vascular, the veins were enlarged and tortuous and the disks elevated 3 diopters; large hemorrhages were noted in the periphery. The patient was usually very stupid. The convulsions began with twitching in the right eyelid.

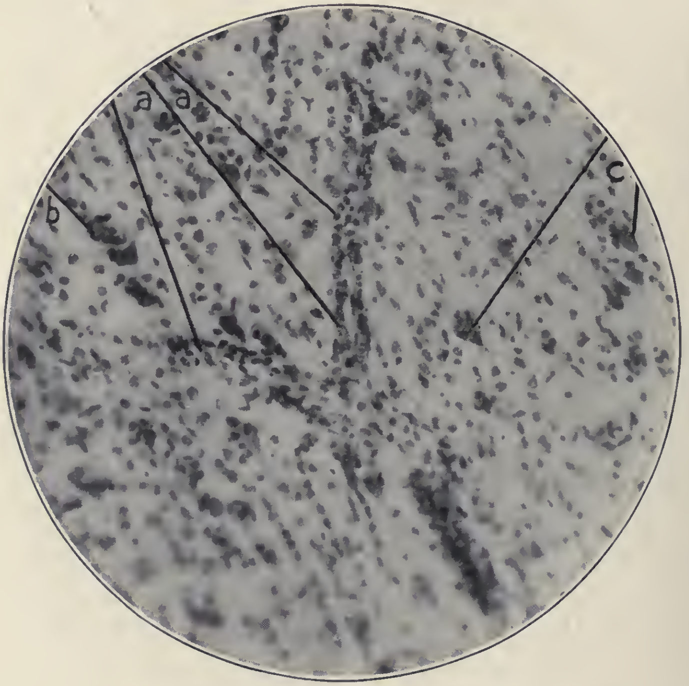

Fig. 2.—Section from the temporal lobe. The center of the picture is occupied by a branching vessel. The vessel walls appear to be infiltrated, but the numerous cells surrounding the branches are glia cells. (Use hand lens.) The rest of the picture is covered by a great number of ganglion cells and glia nuclei. Many of the former are in a state of neuronophagia (*B* and *C*). *A,* glia wall. *B,* ganglion cell surrounded by eleven glia nuclei. *C,* two other ganglion cells invaded by glia cells. (Thionin stain, ×225.) See also Figure 6.

This condition remained unchanged with some slight daily progression for the worse until the child's death, November 4, forty days after operation.

Presumptive Diagnosis.—Basilar tumor (unverified) with hypdrocephalus.

PATHOLOGIC FINDINGS *

Macroscopic.—The necropsy was limited to the brain, which was removed in its meninges after intracarotid injection of formalin. No macroscopic pathologic changes were present except slight hydrocephalus.

Microscopic.—Material: Blocks from the right frontal, motor, parietal, occipital, and temporal regions of the cerebral cortex, left cerebellar hemisphere, and pons, mid-brain, thalamus, and medulla were used. The material was all preserved in 10 per cent. formalin.

Methods: Celloidin, frozen and paraffin sections were stained by thionin, toluidin blue, van Giesen, Weigert's elastica stain, Alzheimer-Mann, Herxheimer's scarlet red stain, and Bielschowsky's silver method.

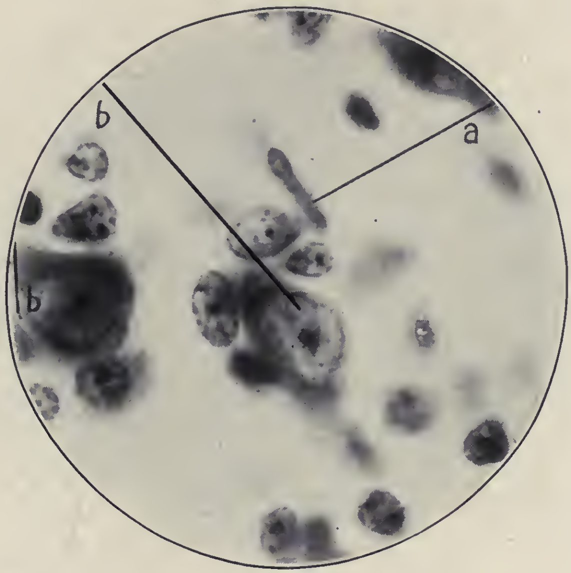

Fig. 3.—Section from the temporal lobe. *A,* rod-cell (Stäbchenzelle). *B,* two ganglion cells in a state of neuronophagia. The rest of the cellular elements are glia cells. (Thionin stain, ×1200.)

A modification of the Alzheimer-Mann (methylblue-eosin) method gave very valuable pictures on formalin-fixed celloidin material. The following procedure was used:

1. Fix in 10 per cent. liquor formaldehydi.
2. Embed in celloidin; cut 15 microns or thinner, in 70 per cent. alcohol.
3. Wash in distilled water.
4. Transfer to aqueous solution of phosphomolybdic acid, from 1 per cent. to saturated, for from one to twelve hours.
5. Pass through two changes of distilled water into

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* From the Pathologic Laboratory of the Cook County and Psychopathic Hospitals, Chicago.

6. Mann's stain (eosin, 1; methyl blue, 1; distilled water, enough to make 100), for from one to twelve hours (preferably twelve).
7. Differentiate directly in 70 per cent. methyl alcohol; move constantly.
8. Transfer to 95 per cent. ethyl alcohol and mount on slide.
9. Add absolute ethyl alcohol on slide.
10. Xylol; Canada balsam (creosote may be used).

The stain comes off in the methyl alcohol in a regular fog and the methyl alcohol differentiation may be carried as far as desired. If left too long in the ethyl alcohol, the eosin will be lost, while the absolute ethyl alcohol also removes the celloidin. The latter contingency may be obviated by adding 20 per cent. chloroform to the absolute alcohol. The mesodermal tissue, namely, the pia and vessels down to the finest capillaries, is stained a bright blue; the glia, violet to purple; the nuclear structures, dark red; the myelin, a bright pink, and the red blood corpuscles, a brilliant orange. The color contrasts are much less striking if examined by artificial light.

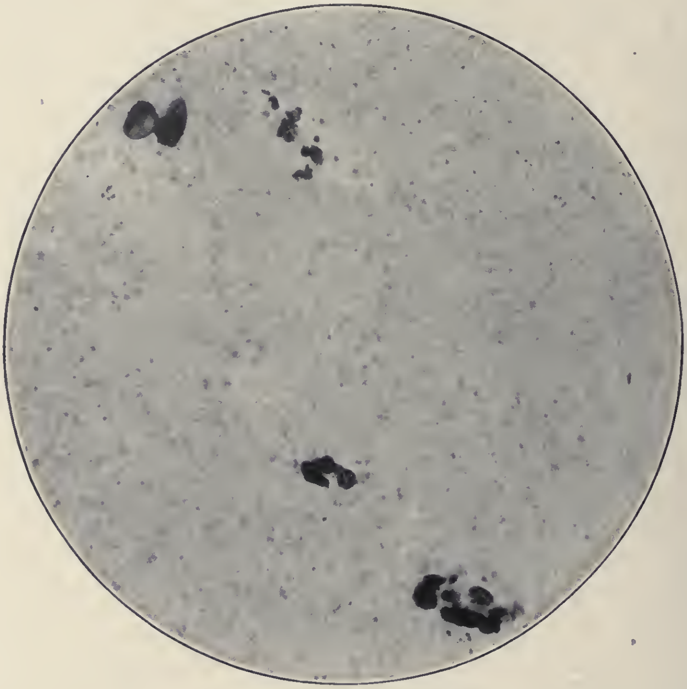

Fig. 4.—Section from the motor area. The perivascular spaces of the vessels are packed with fat (large dark globules). (Frozen section, Herxheimer's scarlet red stain, ×225.)

Meninges: Microscopically the dura showed no changes with the exception of some areas near the longitudinal sinus, consisting of almost structureless formations, containing here and there small lacunae filled each with a single cell (Figure 1). The nature of these masses was hard to determine. They were more or less uniformly stained by thionin, van Giesen, Alzheimer-Mann and Weigert's elastica stain, filled up the enlarged dural interspaces to which they were strictly confined. They resembled cartilage or newly formed bone.

The enlarged subdural space was without cellular or other contents, which, however, were abundant in the pia-arachnoid, subpial (epicerebral) and subarachnoid spaces (Figure 1). The pia-arachnoid cellular infiltration was present everywhere, but was most marked around the medulla, while over the parietal lobe the pia appeared scarcely infiltrated, but edematous.

The localization of the infiltrative elements was best studied in specimens stained by the modified Mann method, and their character was most readily determined in thionin stained specimens. These elements included lymphocytes, plasma cells, macrophages, rod cells, polyblasts, and fibroblasts. Most numerous were plasma cells and lymphocytes, mixed with erythrocytes.

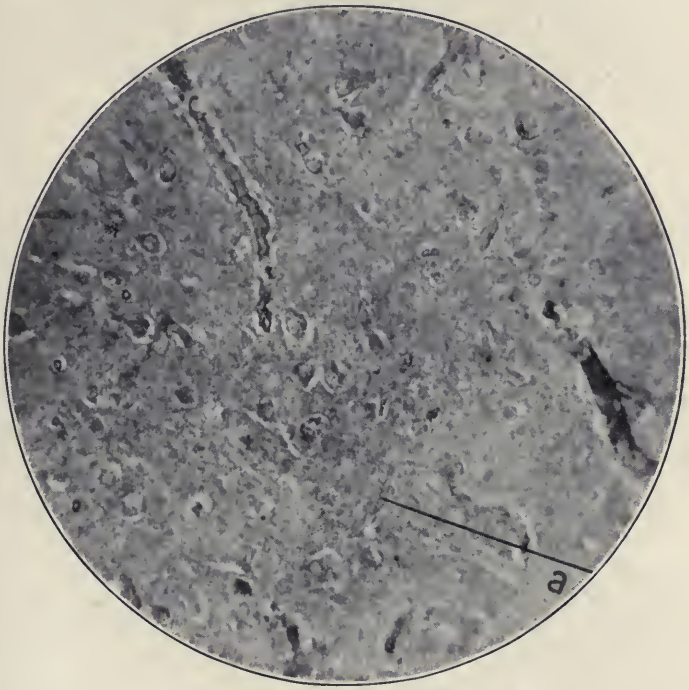

Fig. 5.—Section from the motor area. The field is occupied by a great number of vessels, the majority of which are well developed. In the stained specimen are seen a great number of newly formed vessels, one of which is seen at *A*. (Weigert's elastica stain, ×225.)

In thionin stained specimens, the lymphocytes appeared as small, round, heavily stained, often almost homogeneous nuclei, with no visible cytoplasm. The erythrocytes were cup shaped, pale green bodies without nuclei. Plasma cells were quite numerous, their eccentric nuclei with spokelike chromatin and metachromatically stained cytoplasm with a clear halo alongside the nucleus being clearly discernible. In addition, a considerable number of macrophages were seen packed with blood pigment, which was also scattered freely in the distended meshes of the pia-arachnoid.

The foregoing cellular elements (lymphocytes, plasma cells, erythrocytes, and macrophages) were mixed with so-called mesothelial cells, which are large, round or oblong nuclei containing a scanty amount of chromatin and hardly visible cytoplasm. Somewhat similar were many other nuclei with protoplasm streaming away from either end which may be classified as fibroblasts. Polyblasts with pale horseshoe-shaped nuclei, surrounded with rich cytoplasm, were everywhere in evidence, while around the vessel walls were seen sausage-shaped, oblong nuclei without visible cytoplasm, but rich in chromatin, so-called rod cells (Stäbchenzellen). Polymorphonuclear and cribriform cells (Gitterzellen) were not found.

The vessels of the pia-arachnoid appeared to be distended, but manifested no infiltrative nor other changes.

Cortex: In the cortex, parenchymatous and interstitial changes were in evidence, being most pronounced in the parietal and temporal lobes of the cerebrum; the cerebellar cortex was relatively slightly involved. The parenchymatous changes predominated, and showed only in the ganglion cells, varying in degree from those of Nissl's acute cell disease to total destruction of the cells. Throughout the cortex, the majority of the ganglion cells were very poor in tigroid substance, and the nuclei were rather pale and poor in chromatin. Especially pronounced were these changes in the deeper layers of the cortex, where cell after cell could be seen surrounded by from three to ten robust glia cells (satellitosis). Though present throughout the deeper cortical regions, the satellitosis was especially marked in the parietal lobe, and attained its maximum in the temporal lobe, where it reached to the outermost layers of the

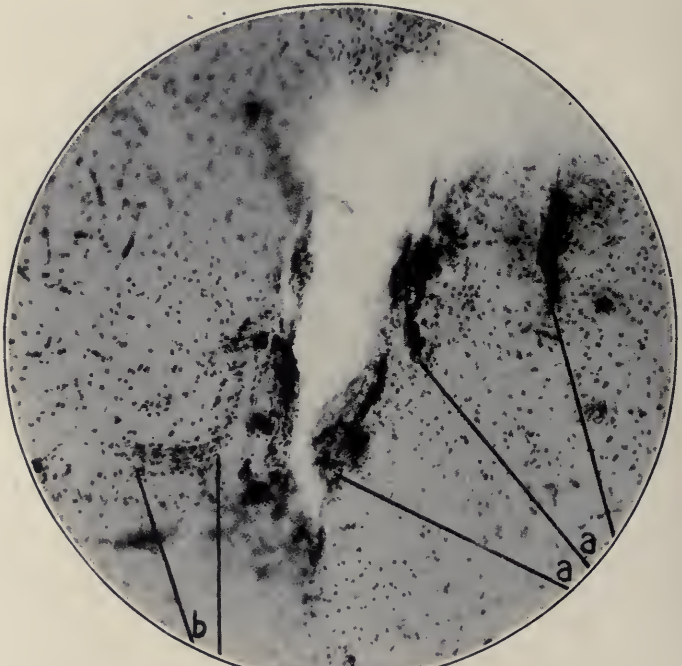

Fig. 6.—Section from the parietal lobe. Some vessels *(A)* greatly engorged and infiltrated. Other vessels *(B)* are not infiltrated, but show a beautiful row of glia cells forming a wall. (Thionin stain, ×82.)

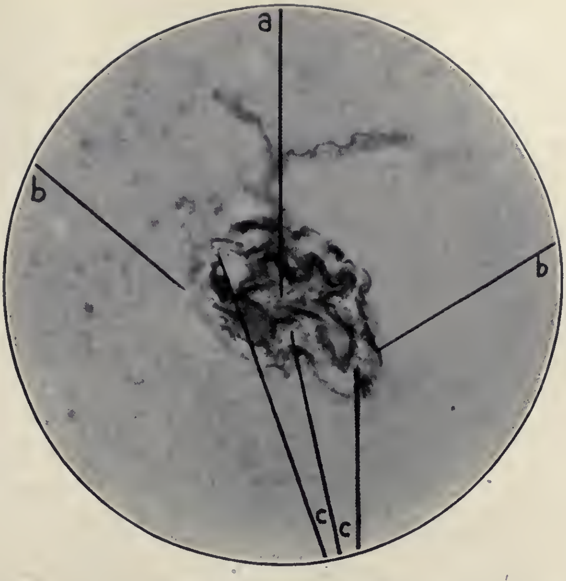

Fig. 7.—Small vein from subcortical area of temporal lobe showing the perivascular space of Virchow-Robin. It contains cellular elements which are not very distinct in the photograph. *A,* lumen. *B;* adventitia. *C,* perivascular space of Virchow-Robin. (Modified Mann's methylblue-eosin stain, celloidin section, ×230.)

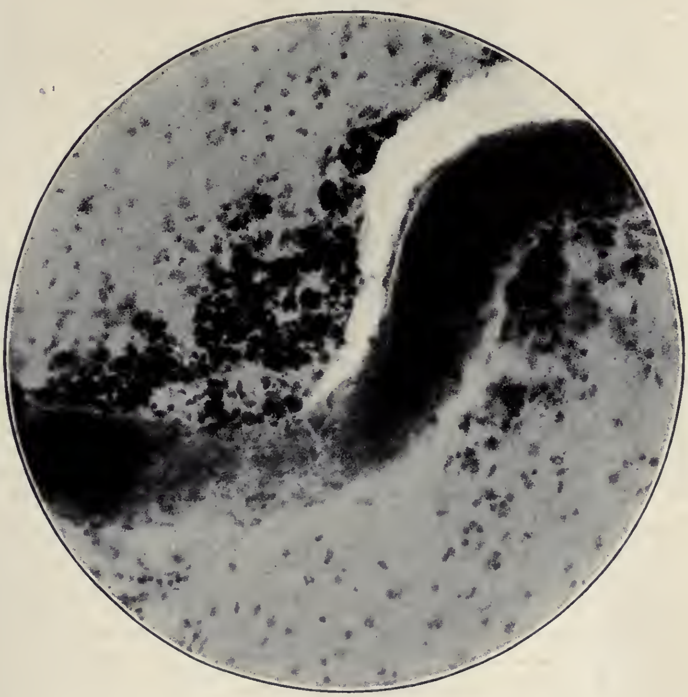

Fig. 8.—Engorged vein from subcortical region of parietal lobe surrounded by an enormous number of macrophages packed with blood pigment (black globules). (Thionin stain, ×225.)

cortex (Figure 2). In these areas, the glia cells not only clustered around but actually invaded the bodies of the ganglion cells (neuronophagia) (Figure 3). The dendrites were shrunken, heavily stained, without Nissl substance and in some places the whole cell was reduced to an amorphous granular mass. Herxheimer specimens showed that these dying cells were packed with fat, especially at the base.

The glia everywhere showed proliferative changes, chiefly in the form of increase in the number and size of cells and chromatin content of the nuclei. Many of the glia cells had an appreciable amount of cytoplasm (protoplasmic glia cells). Around some vessels, especially those where hemorrhage had occurred, a distinct glia wall (Figure 2) was formed—rows of glia cells and proliferated glia fibers. Regressive changes, such as ameboid glia cells, Füllkörperchen, etc., were absent. In the regions where neuronophagia was intense, the glia cells could be seen to contain considerable fat, which was evidently transported to the blood vessels, where fat could be demonstrated in the adventitial cells. Vessels surrounded by such fat-laden cells were seen in all parts of the cortex (Figure 4), but were most numerous in the temporal lobe.

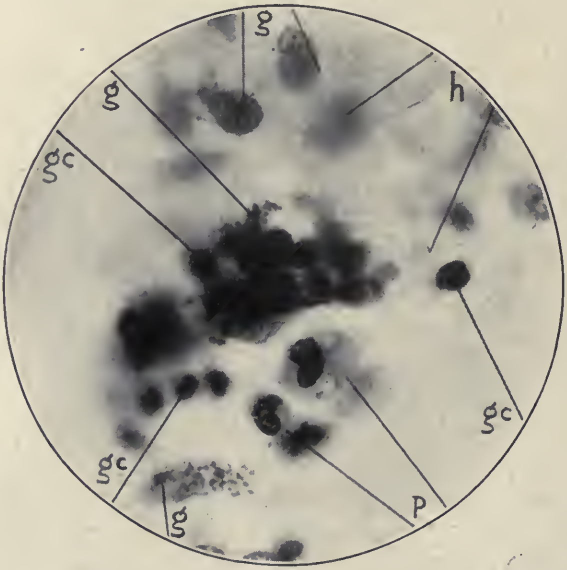

Fig. 9.—Hemorrhage, evidently of recent date. The majority of the glia cells *(G)* are packed with granules of hemosiderin. *H,* hemorrhagic focus. *G,* glia cells containing blood pigment. *GC,* glia cells, out of focus, forming a dense nest in the center and an arch of four underneath. *P,* polyblasts, the nuclei appearing dark because they are out of focus. (Thionin stain, ×1000.)

Interstitial changes in the cortex were not marked, aside from excessive vascularization. Some areas showed an abundance of vessels and capillaries, many of which were very thin and empty, with only a few endothelial cells (Figure 5). Specimens stained with Weigert's elastica stain showed a well developed elastic membrane, sometimes, but very seldom, split into several layers. Inflammatory phenomena were mild, consisting of slight dilatation of the Virchow-Robin spaces, exclusively filled with lymphocytes (Figure 6).

The interstitial changes were especially marked in the subcortical regions. Thus in the temporal lobe there was well developed perivascular infiltration, chiefly with lymphocytes, but numerous plasma cells were also present. Specimens stained with the modified Mann method showed especially well that these infiltrative cells were confined to the Virchow-Robin spaces (Figure 7). Occasionally throughout the subcortical region, but notably in the parietal lobe, a vessel could be seen surrounded by a mass of macrophages engorged with blood pigment (Figure 8), and around these marked glia fiber proliferation. The area shown in Figure 8 is evidently the site of an old hemorrhage and the macrophages are heavily laden with the products of erythrocyte destruction (hemosiderin), but at other places fresher hemorrhages may be found where minute granules can be seen in the masses of glia cells (Fig. 9).

Brain Stem: In the brain stem, the changes described in the foregoing were much less pronounced. In the thalamus was to be found slight perivascular infiltration with lymphocytes and plasma cells. Excessive vascularization was present with newly formed capillaries. Hemorrhages, evidently fresh, without reactive phenomena, were scattered about. Nowhere were there any signs of nerve fiber destruction. Occasionally protoplasmic glia cells were seen, but satellitosis and neuronophagia were practically absent. In the substantia nigra, pons, peduncles, and medulla the changes were similar, but still less pronounced, consisting chiefly of increased vascularity, hyperemia, and occasional fresh hemorrhages. Myelophages, cribriform cells (Gitterzellen) and other pathologic glia changes associated with nerve fiber destruction were totally lacking.

COMMENT

The foregoing combination of interstitial and parenchymatous changes prove that we are dealing here with a case of hemorrhagic leptomeningo-encephalitis, which clinically presented a clear-cut picture of tumor of the left cerebellar hemisphere. More than any case heretofore recorded it deserves the name of "pseudotumor."

There are some extraneous factors in this case which seem to invalidate the diagnosis. For instance, the third nerve palsy was noted as an unusual finding, but was disregarded, because the cerebellar symptomatology was so pronounced, and also because previously it had been seen in cases proved at operation to be cerebellar tumor. Again, the symptoms were stated to follow an attack of influenza, but the father also dated the squint from ivy poisoning! It may be objected that some of the pathologic findings were the result of operative trauma. This is untenable, for the subpial, parenchymatous, and other changes (neuronophagia, perivascular infiltration, etc.) were distinctly of inflammatory infectious nature. Also the pathologic changes were much less evident in the cerebellum at the site of the operation than in the temporal and parietal lobes of the cerebrum, remote from the operative field.

The combination of parenchmatous and interstitial changes will be found in any type of encephalitis, be it dementia paralytica, trypanosomiasis, lethargic encephalitis or poliomyelitis. Yet the present case greatly differs from the foregoing types, especially from lethargic encephalitis. The presence of antemortem hemorrhages, the lack of intensive perivascular infiltrations, and what is more important, the absence of lesions in the substantia nigra, are sufficient to rule out the latter type.

The presence of antemortem hemorrhages might indicate that this was an instance of encephalitis caused by influenza, but in the absence of any reliable histopathologic reports of influenzal encephalitis it would be rash to make such a diagnosis in the present case. The majority of authors[10] (Schroeder, Redlich, and Lhermitte[11]) do not consider the cerebral lesions found in influenza as an encephalitis at all.

In view of the widespread involvement of the brain in cases of meningo-encephalitis, it is not surprising that it is difficult clinically to localize the lesion, nor that striking pathologic lesions should be found in areas not indicated by the clinical findings. In the case reported here the pathologic changes in the cerebellum were adequate to account for the cerebellar symptoms but were overshadowed by more striking changes in the cerebrum.

CONCLUSIONS

1. A case simulating a tumor of the left cerebellar hemisphere proved on microscopic examination to be a meningo-encephalitis Macroscopically the brain showed only slight hydrocephalus.

2. Cases of meningo-encephalitis may run the clinical course of a tumor of the brain even with definite localizing signs but without definite pathologic localization.

3. From a consideration of the cases properly examined, it is certain that no clinical or pathologic entity exists which deserves to be dignified by the term "pseudotumor cerebri."

4. The term "pseudotumor cerebri," if used at all, should be restricted to purposes of clinical classification without attributing to it any etiologic or pathologic significance whatever.

I am greatly indebted to Dr. George B. Hassin for constant advice and criticism, and to Dr. Harvey Cushing for permission to study this case.

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[10] See Bassoe, Peter, and Hassin, G. B.: Contribution to the Histopathology of Epidemic ("Lethargic") Encephalitis, Arch. Neurol. and Psych. **2**:40, 1919.

[11] Lhermitte, J.: L'encéphalite léthargique, Ann. de med. **6**:306, 1919.

DYSTONIA LENTICULARIS (DYSTONIA MUSCULORUM DEFORMANS)*

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The condition generally recognized as dystonia musculorum deformans, a name given it by Oppenheim, is of special interest at this time in view of the recent significant researches concerning the functions of the basal ganglia, especially of the nucleus lenticularis. First separated as a clinical entity by Schwalbe[1] in a Berlin inaugural dissertation in 1908, given a definite nosologic classification by Oppenheim[2] in 1911, and since then the subject of a number of papers and reports, the syndrome, or disease as some would maintain, has recently (1919) received an admirable and comprehensive review at the hands of Kurt Mendel.[3] This last article renders it wholly superfluous to consider in detail the cases hitherto reported, thirty-three in number,[4] according to Mendel, which are abstracted with critical comment by him together with a minute discussion of the condition in its etiologic, symptomatologic and differential-diagnostic aspects. It is my purpose, first, to report two further cases occurring in the same family, one of which is entirely typical and the other doubtful, and secondly to consider certain aspects of this bizarre affection in its broader relations.

REPORT OF CASES

Case 1.—*History.*—Jacob S., of Russian Jewish birth, admitted to the Massachusetts General Hospital, March 13, 1920, was originally referred to the Massachusetts General Hospital from the Peter Bent Brigham Hospital where he had been under the observation of Dr. Harvey Cushing. He was a schoolboy 11 years old, a fourth child of a family of seven. The father was

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* Read at the Forty-Sixth Annual Meeting of the American Neurological Association, New York, June, 1920.

[1] Schwalbe, W.: Eine eigentümliche tonische Krampfform mit hysterischen Symptomen, Inaug. Dissert., Berlin, 1908.

[2] Oppenheim, H.: Ueber eine eigenartige Krampfkrankheit des kindlichen und jugendlichen Alters (Dysbasia lordotica progressiva, Dystonia musculorum deformans), Neurol. Centralbl. **30**:1090, 1911. (Lordosis, "dromedary gait," combination of hyper- and hypotonia important; not a neurosis, four cases.)

[3] Mendel, K.: Torsionsdystonie (Dystonia musculorum deformans; Torsionsspasmus), Monatsch. f. Psychiat. u. Neurol. **46**:309, 1919. (Monographic article with detailed report of previous cases and two additional ones.)

[4] Mendel has evidently overlooked a number of cases reported in America, notably six by J. R. Hunt, published in 1916; see bibliography.

46 years old and the mother 44. Both were living and in general well. The patient had a brother of 19 and a sister of 15, both of whom were well and with no evidence of nervous disorder. One sister, 13 years old, had suffered for seven years from an affection presumably similar to that of the patient. She had been bed-ridden for five years. Another sister of 8 was living and well. The mother had had no miscarriages or stillbirths and denied venereal infection and alcoholism. The birth of the patient was uneventful, and he was apparently normal during infancy. He talked early and walked at 11 months. He had nocturnal enuresis until his seventh year; he had measles in 1912 and whooping cough the following year, with no complications. He began school in his seventh year and made normal progress, getting on well with the teachers but not so well with his companions. A year ago he had had tonsillitis and this year influenza. He had had no other infections or febrile diseases. He had complained of occasional headaches, usually associated with gastro-intestinal disturbances. The pain was usually frontal and temporal, beginning over the right or left eye and spreading backward over the sides of the head. It should, perhaps, be described as discomfort rather than actual pain. There had been no blurring of vision, no deafness or ear discharge. He had had no vertigo, except when nauseated. The cardiorespiratory system had shown nothing abnormal. He had had no dyspnea, precordial pain, cough, sputum, hemoptysis or fever. Several times a year his intestinal tract had been upset, associated with a watery diarrhea. He had had occasional attacks of vomiting. The skin had shown a tendency to sweating in those parts which were in muscular movement. He had had no eruptions or loss of hair. His weight had varied between 50 and 60 pounds. His habits as regards food, hours of meals and general hygiene had been, on the whole, good.

The disorder of motion began in July, 1916, with slight spasm of the right arm. He began to use his left arm instead of his right. The spasm was most marked when he attempted to use a pencil. The arm appeared weak, but he did not drop objects. The spasm gradually spread to the shoulder and then began to involve the left shoulder, passing finally to the arm. Two years later his left leg began to be affected by muscular spasm and last winter his right leg was affected in a similar way although he was still able to walk. These spasmodic movements, described later, became progressively worse so that since February, 1920, he had been unable to go out alone though he still managed to get about somewhat in the house.

Physical Examination.—This revealed a normal child except for the motor disorder which entirely dominated the clinical picture. He was sufficiently well developed and mentally was bright and responsive. He talked well and showed an intelligent interest in the situation quite beyond his years. The skin, mucous membranes, chest, heart, lungs, genital organs and abdomen, showed absolutely no abnormality. The blood pressure, systolic was 105, and diastolic, 60.

The electrical examination of the muscles gave normal faradic and galvanic response (left arm tested) both direct and indirect. The galvanic response was quick with no indication whatever of muscle or nerve degeneration. The white blood cells numbered 6,800; differential count: polymorphonuclears 74 per cent., lymphocytes 24 per cent., large mononuclears 2 per cent. The red cells and platelets were normal; hemoglobin 75 per cent.; fasting sugar 0.07 per cent.; blood calcium 12.2 mg. per 100 c.c. of plasma. The guaiac test was normal. The urine on three examinations showed varying specific gravity from 1.010 to 1.030, without sugar, albumin or other abnormal constituents.

Examination of the Nervous System.—This revealed unequal pupils, the left larger than the right; normal light and accommodation reaction; visual fields normal; slight nystagmoid movements on lateral fixation, not on direct fixation; no diplopia or paralysis of ocular muscles; fundi normal; Rinné test positive and in general hearing not affected; facial nerve normal. The fifth nerve was normal both in its sensory and motor distribution. The tongue protruded in a straight line; it was not coated. The teeth were imperfect; they were badly neglected but well placed and of good form. The palatal arch was low. The tonsils were small; palatal reflexes normal; there was no difficulty in deglutition. The arm reflexes were difficult to obtain on account of constant involuntary movements; they were not increased. The dynamometer showed the right hand grasp weaker than the left. There was no pain over the nerve trunks, no astereognosis, loss of position sense or other disturbance of sensation. The abdominal reflexes were present and equal; the cremasterics were not obtained. Knee jerks were equal and normal. There was no ankle clonus and no Babinski response. Here also the exact determination of the reflex condition was difficult to obtain on account of continuous muscular spasm. (It should be said that observations made at different times by different observers gave somewhat different results. It is, however, entirely fair to assume that the reflex conditions are essentially unchanged.) There was no muscular atrophy. The boy was of slender build but beyond the fact that some of the muscles were small they could not be regarded as in any sense abnormal. A conspicuous feature was variation in muscular tone. Some muscles were firm and elastic, showing their contours, others were flabby and toneless. In the former group were in general included the leg muscles with the exception of the tibials and peronei; both the flexors of the knee and the opposed extensors were tonic. The extensors of the hip seemed to be in greater activity than their antagonists. The triceps were tonic and opposed to the atonic biceps. The flexors of the wrist tended to overcome the extensors and at the shoulder both the deltoid and pectoralis major were tonic. The left sternomastoid was more active than the right. These observations concern the boy's usual position but as soon as he moved these balances were upset and the extraordinary spasm developed until he again placed himself in a position of rest. The foregoing description of the muscle tone can, however, not be regarded as constant even in the resting state. The facial muscles showed no spasm or contortion; in fact the muscles supplied by the cranial nerves were essentially uninvolved in the general spasm. Occasionally there was a sharp contraction of the sternomastoid. Usually when the patient was examined lying quietly in bed there was no movement of the body but the right arm was held constantly in extension and extreme inward rotation with wrist and fingers sharply flexed. This arm for certain periods might be relaxed, but there was a constant tendency to return to the contracted position. In attempting to touch his nose with the index finger of the right hand the arm was flung forward over the chest with extended forearm, then the arm was violently flexed, finally reaching the nose. This movement could only be made at a high rate of speed; a slow movement never attained its object, the arm remaining extended across the chest. Not infrequently it was impossible under any conditions to flex the arm at the elbow on account of the spasm and the attempt to reach the nose was then given up. On attempting to make a movement, as for example, sitting up in bed, the head was thrown violently to one side and backward with extreme anteroposterior flexion of the spine; one shoulder was raised above the other and fruitless attempts were made to flex the arm at the elbow. The fingers were in the meantime in a condition of flexed spasm. As he sat up in bed with his back bent as described, the legs were flexed at the hip and the lower legs very sharply flexed against the thighs. There was no marked spasm of the feet. The head was often thrown to the right with a lateral curvature of the spine. In any attempt to move the asynergy was greatly increased. There was no hypertrophy or, as stated above, atrophy of the muscles.

Gait.—The gait of the patient was beyond adequate description. He forged forward with the pelvis sharply tilted, head and chest thrown violently back in a position of opisthotonos making extreme incoordinate, choreiform or athetoid movements to retain his balance and progress. This apparently was only possible under certain circumstances by twisting the body violently, to which movement the name torsion spasm has appropriately been given. He thrust one leg far forward and twisted himself, as it were, into a further position of equilibrium. Oftentimes in accomplishing this progression the patient turned half or even completely around and took a step backward. In fact, as has been described in other cases, progression backward may be accomplished in a much more orderly fashion than forward movement. The effort with which any progress is made is exhausting so that after an attempt to walk 20 feet, more or less, the patient is ready to drop from fatigue. The patient was able to get up from a lying position on the floor without the slightest difficulty so far as muscular strength was concerned. He showed none of the weakness or climbing efforts characteristic of the dystrophic patient but assumed the erect position with extraordinary rapidity and with various spasmodic and incoordinate movements such as characterized all of his motor acts.

Summary.—A boy of 11, of Russian Jewish parentage, was well until his seventh year; then there was gradual development of a spasmodic movement of the right arm, shoulder, left shoulder and left arm with subsequent involvement of the left leg and within a year of the right leg. Muscular movements were characterized by spasm alternating with relaxation giving rise to a gait characterized by torsion movements of extreme degree. Other than the motor disturbance there were no physical or mental abnormalities.

CASE 2.—*History.*—Ida S.,[5] sister of the foregoing patient, aged 13, was admitted to the Peter Bent Brigham Hospital on Feb. 29, 1920, for gangrene of the foot. She was born in Chelsea, Mass., the third child in a family of five. The birth was normal and she developed without incident, walked at 1 year of age, talked at 2 years and was in no way abnormal up to the age of 6. She had had tonsillitis frequently until she was 7, when the tonsils were removed; she had whooping cough at 3, measles and scarlet fever at 11. In her seventh year what was regarded as "chorea" developed, manifesting itself first as a twitching of the neck and gradually spreading to the limbs and body. From this time on she also showed definite mental impairment and general retardation of body growth and had practically been confined to bed since her seventh year. For over two years she had not been able to talk intelligibly. From time to time she had had diarrhea with elevation of temperature .

Physical Examination.—This revealed a girl of 13 with the development of a child of 8 or 10. The most obvious disturbance was a constant choreiform movement extending over the entire body and also involving the extremities. This was not continuous but the patient remained in any one position only for a brief time. The facial expression was drawn and the muscles of the face were definitely involved in the choreic movements. She made only inarticulate sounds and appeared to be markedly retarded mentally. The head was very large for the size of the body, tended to be square in shape and had a moderate amount of coarse, brownish red hair. The scalp was dirty and there were pustular eruptions over the back of the head and right side of the face. The pupils were equal and reacted to light; the accommodation could not be determined. There was some opacity of each cornea which, however, apparently did not markedly obscure the field of vision. The patient's eyes shifted constantly from side to side to such an extent that it was impossible to determine whether or not a strabismus existed. The ears showed no abnormality. The nose was normal. The patient could open the mouth only by making extraordinary facial contortions, and she was unable to protrude the tongue. The thyroid was not enlarged. The thorax was infantile in type but expansion was good and equal on both sides. The heart showed nothing of significance beyond a systolic murmur at the base. The pulses were equal, regular, synchronous and of fair volume and tension. The systolic blood pressure was 90; the diastolic, 60. The lungs showed no involvement. The abdomen was protuberant. There was marked dilatation of the superficial veins over the abdominal wall. The liver, spleen and kidneys were not palpable. There were no masses or muscular spasm. Both arms and legs were held in a flexed position preventing full extension even by force. Both feet showed talipes equinus. The toes and fingers were retracted in flexion. Over the right toes and extending on to the dorsum of the foot there was a definite gangrene, presumably due to careless treatment before admission to the hospital. The emaciation of the patient was extreme. Her appetite, however, was good but she was extremely constipated. She had no nausea or vomiting and there was nothing definitely wrong with her gastro-intestinal tract. She was incontinent, presumably because of her mental state and her incapacity to communicate her needs. She slept only by the aid of sedatives which she had been taking for about two years. It was noted that the movements almost ceased during sleep but not wholly. The urine on admission gave a high specific gravity, 1.032; acid reaction, normal color, no albumin or sugar. The hemoglobin was 60 per cent. and the white blood count 6,040.

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[5] I am indebted to Dr. G. Horrax of the Peter Bent Brigham Hospital for this history.

She was discharged March 31, 1920, after a residence in the hospital of about one month. During that time she was noisy, difficult to manage, disturbed the other patients in the ward and in general showed definite mental enfeeblement. The gangrene of the foot yielded to treatment and she was returned somewhat improved to her home.[6]

Summary.—The essential features in this case are apparently normal development up to the age of 7, then the appearance of widespread choreiform movements which uninterruptedly persisted up to the time of her death, a period of about six years. Mental failure, disturbance in speech, more or less facial contortion, extreme weakness and emaciation accompanied the choreic movements. In general, the physical examination, with the exception of intercurrent disorders, was essentially negative.

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[6] This patient has since died, unfortunately under conditions which precluded a postmortem examination.

The cases are primarily of interest since they occurred in the same family, and secondarily because they are totally dissimilar in clinical manifestations. Both patients were attacked by the disorder at about the seventh year, from which one has since died. The disorder of motion in Case 1 has never involved the head, although, contrary to the description of many cases, it began in the upper extremities, whereas in Case 2, the neck was affected at the outset, and until her death, the illness was characterized by involvement of the head. The boy has retained his mental capacity to the fullest extent, and in fact shows no signs or symptoms referable to the head or cranial nerves except a slight pupil inequality, whereas the girl forthwith became demented. It is altogether probable that the two cases are familial in the sense that they imply some common defect of development but the second case cannot properly be classified as dystonia musculorum deformans as that term has come to be used. The first is a classic example of the condition described and named by Oppenheim, whereas the second represents, doubtless, a lesion in the same general area of the brain but with entirely different manifestations, a matter of importance in our final classification of the disorder. That two or more cases of a relatively definite and similar type may occur in one family is shown by the cases recently described by Abrahamson,[7] in which he had the opportunity of observing three members of one family, all presenting a disorder of motion of characteristic dystonia type. It remains clear, however, that with few exceptions the cases hitherto studied have occurred sporadically in much greater degree for example, than in Friedreich's ataxia, periodic family paralysis, or certain other definite familial diseases.

TERMINOLOGY

It is inevitable, but none the less unfortunate, that in the effort to establish the condition as a clinical unity, a variety of terms have been used, which further study has shown to be inadequate, if not wholly misleading. Schwalbe, in the first publication on the subject, described the condition as "an unusual variety of spasm with hysterical symptoms." This has the merit of vagueness, but it is altogether doubtful whether hysterical symptoms occur in the clinical picture. Ziehen[8] speaks of it as a "tonic torsion neurosis," which takes no account of the characteristic atonia, and also assumes by the use of the word neurosis that it has no structural basis. Oppenheim's terms

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[7] Abrahamson, I: A Case of Dystonia Musculorum Deformans (Oppenheim), J. Nerv. & Ment. Dis. **40**:38, 1913; ibid. **57**:451, 1920.

[8] Ziehen: Fall von tonischer Torsions-neurose, Neurol. Centralbl. **30**:109, 1911.

"dysbasia lordotica progressiva" and "dystonia musculorum deformans" are less open to objection. Of these the latter terminology is to be preferred, but the use of the word "deformans" is misleading and inaccurate. Flatau and Sterling[9] add nothing in their "progressive torsion spasm in children," since the important element of dystonia is ignored and presumably also the affection is not confined to children. Fraenkel[10] considered the disorder allied to tics and spasmophilia and used the phrase "tortipelvis" which emphasizes only one and that an inconstant element. "Athetosis universalis," as used by Förster, is entirely inadequate since the movements are not universal and not truly athetoid in character. Very much better terms, because more general, are those used by Strümpell, "amyostatic symptom complex," or "myastasia;" by Mendel, "torsion dystonia" and by Thomalla "dystonia lenticularis." Of these Thomalla's[11] term is, on the whole, to be preferred since it describes adequately the muscular condition and suggests its anatomic location. Until our knowledge is much more exact than at present, this is as far as it is safe to go.

ESSENTIAL CHARACTER OF THE CONDITION

As one would expect from the diversity of terminology, the effort has been made to establish the disorder as a definite clinical entity. Oppenheim was inclined to this view and recently Mendel has asserted with conviction that we are entirely justified in including the cases in a special disease group and in postulating for it a specific etiology, a "morbus sui generis." Hunt,[12] Jelliffe, Thomalla and Strümpell are opposed to any such narrowing of our point of view which tends to divert investigation from the larger questions at issue. Experience has amply shown that a syndrome, without a defined pathologic anatomy and still less an unascertained etiology, should never be regarded as a "disease entity." The history of medicine is replete with errors into which a failure to recognize this fact has led. In this particular instance a review of the cases described as "dystonia

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9. Flatau, E., and Sterling, W.: Progressiver Torsionsspasmus bei Kindern, Ztschr. f. d. ges. Neurol. u. Psychiat. **7**:586, 1911. (Jewish children; location: peduncles, cerebellum.)

10. Fraenkel, J.: Dysbasia lordotica progressiva; Dystonia musculorum deformans; Tortipelvis, J. Nerv. & Ment. Dis. **39**:361, 1912. (Analogy with torticollis; develops on a "tic" soil. Deficit in calcium metabolism.)

11. Thomalla, C.: Ein Fall von Torsionsspasmus mit Sektionsbefung und seine Beziehungen zur Athétose double Wilsonschen Krankheit und Pseudosklerose, Ztschr. f. d. ges. Neurol. u. Psychiat. **41**:311, 1918.

12. Hunt, J. R.: The Progressive Torsion Spasm of Childhood (Dystonia musculorum deformans), J. A. M. A. **67**:1430, 1916. (Six cases reported; valuable paper, overlooked by Mendel.)

musculorum deformans" forthwith shows the divergence in the symptomatology. A great variety of irregular muscular movements have already been described under the general category; torsion, tortipelvis, dromedary gait, athetosis, choreiform movements, tremor, propulsion, atonia, hypertonia, but none, with the possible exception of a general dystonia, can be regarded as sufficiently characteristic to meet the requirements. It is increasingly evident that to postulate this syndrome as a disease is to lose sight of the main issue, which is to determine under what conditions of altered structure or function such abnormal movements arise. It is therefore desirable to give up for the present the attempt to circumscribe the disorder further than to regard it as one of the outstanding symptom groups of lenticular or basal ganglion disease. The great numbers of transitional forms already described sufficiently demonstrate that it has small claim to clinical independence. A provisional classification is all that should be undertaken for the present, an attempt which Cecile and Oskar Vogt[13] in their paper on the Pathologic Changes of the Striatum and Pallidum have recently attempted. Little light has as yet been thrown on the condition through post-mortem examination. The changes in progressive lenticular degeneration have been adequately described by Wilson[14] and many subsequent observers. Von Economo[15] has recently presented a detailed account of the gross and histologic alterations but no unquestioned case of dystonia musculorum has yet been studied, unless we except one reported by Thomalla.[16] Even this case should be regarded as atypical for the following reasons, as Thomalla himself points out: the Babinski response was not always negative; there were associated athetoid movements. Necropsy showed marked changes in the putamen, which was reduced in size on both sides and softened. Microscopically the parenchyma of the putamen was fully destroyed. There was definite glia reaction in the meshes of which fat granule cells appeared. The blood vessels showed minor changes. The caudatus, globus pallidus and internal capsule were not involved. The thyroid was somewhat enlarged; there were doubtful changes in the testicles and most important, alterations of the liver, similar to those described in Wilson's disease, were well defined. Regarding the liver changes, Thomalla believes that they represent a fundamental defect of development, in the basis of which a progressive degenerative process has occurred associated with secondary connective tissue proliferation.[17] Finally Thomalla concludes that bilateral athetosis, pseudosclerosis (Westphal-Strümpell), Wilson's disease and torsion spasm (dystonia musculorum) all characteristically show disturbance in muscle tone, and judging from the one necropsy of a case which clinically bore some resemblance to the latter condition, may be supposed to have a similar pathologic anatomy. For all of these conditions the appropriate term of "dystonia lenticularis" is proposed.

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[13] Vogt, C., and O.: Sitzungsberichte der Heidelberger Akademie der Weissenschaften, No. 14, 1919.

[14] Wilson, S. A. Kinnier: Lenticular Degeneration, A Familial Nervous Disease Associated with Cirrhosis of the Liver, Brain **34**:295, Part 4 (March) 1912.

[15] Von Economo: Wilson's Krankheit und das Syndrome du Corps Strié, Ztschr. f. d. ges. Neurol. u. Psychiat. **43**:173, 1918.

[16] One of Ziehen's original patients is said to have come to necropsy with negative findings (reference not found) and more recently one of Flatau and Sterling's cases, which showed the Wilson type of degeneration in liver and putamen, although the patient clinically appeared to be a victim of dystonia musculorum (Jelliffe).

Strümpell takes a similar position in the use of the term "amyostatic symptom-complex" or "myastasia." Under this general term he includes extrapyramidal lesions, namely: Wilson's disease, pseudosclerosis, double athetosis, paralysis agitans, juvenile paralysis agitans and presumably dystonia musculorum. He believes it even possible to group in the same general category his pseudosclerosis and Wilson's progressive lenticular degeneration as exogenous disorders characterized clinically by tremor of wide range, disturbance of speech, dementia, pigmentation of the cornea (not observed by Wilson), often muscular rigidity with alterations of the liver (not primarily observed by Strümpell) and marked degeneration of the lenticular nucleus. The analogy with the condition clinically termed paralysis agitans naturally assumes importance.

In a recent discussion of Abrahamson's communication, to which reference has already been made, Jelliffe outlines the only tenable position which it seems possible to hold in the present state of our knowledge when he states that dystonia musculorum is simply one of the striking types of the varying clinical trend of the group of striatum syndromes, which includes also Vogt's double athetosis, Westphal-Strümpell's pseudosclerosis, Wilson's lenticular degeneration, Huntington's chorea, tuberous sclerotic idiocy and paralysis agitans. Jelliffe also insists that a matter of prime importance, as yet in its speculative state, is the association between liver and striatum changes with the possibility, which the future holds out, of arriving at some sort of an understanding of the vegetative control of the muscle metabolism through the possible striatum connections.

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[17] Little attention has been given to the possible rôle of the cerebellum in relation to the condition of dystonia. Flatau speaks of the possibility of the peduncles and the cerebellum being involved, but no systematic attempt has been made, either theoretically or postmortem to determine this point, in spite of the recognized part played by the cerebellum in the maintenance of muscle tone and coordination.

CONCLUSIONS

The final question of diagnosis of dystonia musculorum loses much of its force if we are to consider it as a symptom group merely and not as an entity, as Mendel among others insists. As the number of reported cases grows it becomes increasingly evident that transitional forms are far outnumbering those which conform to an assumed definite type with the natural result that nothing is gained by an attempt to establish its claim as a separate "disease entity." Certain conclusions are, however, justified.

1. A type of motor disorder exists sufficiently definite to demand a provisional classification, characterized by combined hypertonia and hypotonia (dystonia) giving rise to a distinctive form of gait and motor disturbance in general.

2. Although occurring with frequency in children of the Jewish race, it is not confined to that race, nor does it always begin in childhood.

3. It is sporadic, rather than truly familial, though in a few instances more than one case has appeared in the same family (Schwalbe, Bernstein, Abrahamson, Taylor, one case atypical).

4. It is not to be sharply differentiated from various other forms of motor disorder of presumable extrapyramidal origin.

5. It is due to structural alterations of the striatum (lenticular nucleus).

6. It has no defined pathologic anatomy, but its association with alterations of the liver is probable.

7. The etiology remains wholly obscure beyond the probability of congenital defect.

8. For the present, the most suitable term both in its clinical and pathologic implications is dystonia lenticularis.[18]

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[18] Additional references:

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Higier, H.: Ein Fall von Dystonia musculorum progressiva deformans, Neurol. Polske. **2**:45, 1911.

Keschner, M.: Dystonia musculorum deformans, J. Nerv. & Ment. Dis. **47**: 103, 1918.

Maas, O.: Zur Kenntnis des Verlaufs der Dystonia musculorum deformans, Neurol. Centralbl. **37**:199, 1918. Discussion, idem., p. 221. (One patient 49 years old; face involved; relation to pseudosclerosis; Wilson's disease and paralysis agitans; may have psychic symptoms; may improve. Oppenheim doubts the case, Kramer disagrees.)

Patrick, H. T.: Dystonia musculorum or Tic, J. Nerv. & Mental Dis. **44**: 63, 1916. (Age 27; not Jewish; face and upper extremities involved; question of torticollis; two cases reported. Bassoe reported a possible case in discussion.)

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Spiller, W. G.: A Case of Dystonia musculorum deformans, J. Nerv. & Ment. Dis. **40**:529, 1913. (Middle-aged man, probably not Jewish; three cases in family, one reported.)

Sterling, W.: Ein Fall von progressiven Torsionspasmus, Gaz. Lekarska., No. 21, 1916.

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THE CHRONICITY IN LETHARGIC ENCEPHALITIS *

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During the past eighteen months over two hundred cases of lethargic encephalitis have come under our care, chiefly in Mount Sinai Hospital and in consultation at the Jewish Hospital, Brooklyn. The febrile stage of most of these patients lasted from ten to twenty days. When that stage was passed and convalescence was well established, they were discharged from the hospital. As a visiting physician watching the passage of these patients through our intern service, I at first thought lethargic encephalitis was an infectious disease of sudden onset and of rapid course which, if terminating happily, would be followed by a less rapid but complete recovery. The cases of some patients undoubtedly conformed to this view; others, in which the acute stage differed in no way, halted in their recovery and were forced to return to the Outpatient Department for further treatment. Still other patients survived the acute phase only to enter a chronic progressive stage. With every month that passed, more of these unfortunates returned. The early optimism I enjoyed quickly perished, and I learned to dread this disease, so often fatal, not infrequently inflicting permanent damage on those who survived it, and sometimes bringing in its train progressive functional deterioration.

No one is qualified from personal experience to give a definitive characterization of lethargic encephalitis, and least am I, for my experience with this disease has been gained chiefly among the racial group of the Jewish community of New York City and its neighborhood. Each of us has seen his little section of the epidemic and from it has drawn his own conclusions, which are neither the first nor the last word, but merely a small part of the general truth. It is, therefore, undesirable at this stage of our ignorance to risk a permanent distortion of our view of the disease by unduly stressing either local or personal experience. Hence, without pretence to completeness and without attempting statistical presentation, I shall concern myself merely with a brief description of some of the chronic types and persisting consequences of lethargic encephalitis.

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THREE ELEMENTS IN LETHARGIC ENCEPHALITIS

We distinguish three elements in the disease: (1) the disease agents; (2) the effect of these agents on invaded tissues, and (3) the physiologic and psychologic consequences of these morbid effects. We know that the agents sometimes actively persist for periods far exceeding the usual twenty day limit. We know further that these disease agents sometimes produce irreparable destruction in nerve and vascular tissues and perhaps in the glandular tissues. Such permanent structural changes, in so far as they are definitive and invariable, can scarcely be classified as chronic; for the word chronic is used habitually to connote not merely an enduring, but also an active, change or process. Nevertheless, permanent mechanical defects in the structure of the body, although themselves invariable, may be followed by progressive functional deterioration to which the term chronic can be applied. And, moreover, the deterioration of a function, even if not itself progressive, may entrain a gradual disorganization of an associated function. Hence, the chronicity of lethargic encephalitis may have its origin in any one of the three elements I have distinguished: in the disease agents, in the structural alterations these produce, and in the physiologic consequences of these alterations.

Disease Agent.—The persisting disease agent may give rise to a continuous or remitting fever. The point at which a protracted infection merges into a chronic infection, can be determined only by the application of arbitrary time standards, about which at present there is no conventional agreement. The fever is probably due to the tissue response to the organism itself. When the fever finally subsides organisms may still survive in sheltered sites, where, acting with greatly reduced virulence, they may insidiously produce progressive lesions; or, if the organism itself becomes emasculated, so that it lacks the power to destroy or to injure tissues, a toxicity may still persist of a potency sufficient to cause gradual cellular damage or death and to excite in and around the affected cells the repair reactions consequent to such accidents. For instance, Buzzard has shown that in late cases of lethargic encephalitis atheromatous changes occur in the blood vessels of the central nervous system. The deposit of lime salts does not occur in healthy tissues. In the cells in which lime salts are found, degeneration has preceded its deposit. The calcification is, so to speak, compensatory to failure of the natural repair processes of the degenerated cells. We do not definitely know, however, whether the toxin which produces the vascular degeneration is derived directly or indirectly from the organism of lethargic encephalitis. As the endocrine glands are implicated in this disease, many abnormal chemical changes accompany it. These chemical aberrations affect all the cells of the body, but the calcareous degeneration is found chiefly, if not solely, in the blood vessels of the central nervous system. It is, therefore, unlikely that autogenous chemical changes are alone responsible for the slowly developing vascular defects. In other words, it is at least probable that there exists a chronic and toxic type of the disease.

Structural Alterations.—The structural changes which endure occur in the highly organized cells—nerve, vascular and glandular cells. In the present state of our knowledge, we deduce the nature of the anatomic changes in these cells only from the alteration in the physiologic conduct. Some of these physiologic alterations are not permanent. Their disappearance presumably depends either on the absorption of inflammatory exudates or on the gradual extinction of the disease agents, permitting the functional recovery of cells which were not vitally damaged. As our observation of these physiologic changes has lasted at most only eighteen months, it is possible that changes which we might now deem permanent may yet disappear; that changes which now seem stationary may later become progressive; and that changes which are now progressing may hereafter become stationary or even retrogress. In other words, we are not yet in a position to undertake the defining and classifying of chronic types of lethargic encephalitis. All that our present knowledge justifies is the recording of the signs that tend to persist. I have collected these persistent signs, and for convenience present them in the groups, somatic, endocrine and mental.

Physiologic and Psychologic Consequences of Alterations. — The persistent somatic signs are fatigability, diplopia, blurred vision, optic neuritis and optic atrophy, faulty accommodation, nystagmoid movements in extreme lateral positions and slight differences in the width of the palpebral apertures, lack of facial expression, tongue tremors and deviations, peripheral palsies, a pseudo-Parkinson syndrome, fine and coarse tremors of the extremities, a hemiplegic spastic attitude of the upper extremity, dragging of the leg and increased resistance to passive movements with exaggerated deep reflexes, propulsion and retropulsion, unsteadiness and titubation, careful, short or unequal steps and other disturbances of gait and attitude, chorea, athetosis, dystonia, epilepsy, aphasia, all varieties of sensory disturbances and a transverse myelitis syndrome which may lead to death from decubitus or pyelonephritis.

Among the endocrine signs are obesity, menstrual irregularity, especially amenorrhea, Sergent lines, dermography, von Graefe, Moebius and similar eye signs, hyperhidrosis and anhidrosis, polyuria, low blood pressure, persistent slow pulse and tachycardia, either constant or paroxysmal, labored breathing and asthma, impotence and medullary crises.

Among the mental symptoms, those observed during the acute stage of the disease may long persist in a mitigated form. If the disease has been acute, especially if meningo-encephalitis signs occurred, the patient may remain as if stunned for months. Frequently an amnesia exists during part of or the whole period of the disease. It may even be retro-active. Some patients confabulate to fill in the memory gaps, especially such as developed a confusional psychosis of the Korsakoff type. Patients are seldom desirous of leaving the hospital. When they return to their homes even those who were quiet while in the wards may become peevish and complaining, so that often the family begs us to take the patient back. This irritability may be combined with wilfulness and anxiety. Part of this mental state arises from the belief of the patient and of his friends that death has been evaded only by a miracle. Sometimes depressions and hypomanias develop; at other times hebephrenic episodes are seen; and even fully elaborated psychoses may appear, which the family or personal history leads one to suspect are due to hereditary tendencies which the infection has brought out. Many weeks or even months usually elapse before these patients return to work, for they are timid, fearful and tired, and unable to apply themselves. To their incapacity is added a marked emotionalism. They linger in bed far into the day, and when they arise they sit about unoccupied and vaguely restless. Some are definitely hysterical; many acutely neurasthenic. The uncontrollable laughing and crying of the convalescent case of lethargic encephalitis recalls the emotional instability seen in multiple sclerosis and in pseudobulbar palsy.

Children show the same mental defects as adults. Some, however, in whom the disease began with insomnia, for months remain drowsy by day but wakeful and irritable at night.

Besides these mild forms, severe mental disturbances may persist or develop. I have seen both grave delusional and confusional psychoses, as well as a form of amentia resembling dementia paralytica (except that grandiose ideas are absent).

In this brief enumeration of such symptoms and signs of lethargic encephalitis as are remarkable for their persistence, I have not attempted to separate the stationary from the progressive, or the persistent from the permanent. In some cases, although the lesion may be permanent, the physiologic consequence may doubtless be adequately compensated and the functional disturbance may disappear. In other cases compensation may be imperfect, or it may be lacking and a constant and invariable functional disturbance persist. And if, as in the case of the internal secretions, the affected function belongs to a confederacy of functions, the whole confederacy may be deranged and thus indirectly a progressively increasing defect develop. This effect of implication of associated functions is best seen, not in the physiologic but in the psychologic, disturbance. Heredity, environment and age may enhance or diminish the mental defect and may determine its development, its persistence or its disappearance. The factors in the chronicity of the disturbance of lethargic encephalitis are thus not merely the disease agent and the resistance of the person to this organismal invasion, but also the general functional integrity of the individual and the nature of his environment.

CHRONIC AND PROGRESSIVE FORMS OF LETHARGIC ENCEPHALITIS

Cases of protracted and imperfect recovery, as well as cases in which the deterioration of particular functions progresses, are in many instances to be considered not as chronic types of lethargic encephalitis, but as examples of the effect of the disease on persons of certain types or circumstance. In some instances such cases undoubtedly merit classification as chronic types. Thus, we may distinguish as a separate type the group of chronic cases which resemble Parkinson's disease. Another type comprises the chronic choreas; still another, the progressive dystonias and a fourth, the cases of progressive myelitis which in my experience usually end fatally. Other chronic and progressive forms may have been observed which are not within my experience. I shall not attempt to describe these four types, and I shall only mention without discussing the noteworthy fact that multiple sclerosis is not a sequel to lethargic encephalitis.

Editorial

RECENT ADVANCES IN NEUROLOGY OF THE PERIPHERAL NERVES

The late war provided an inexhaustible fund of experience in neuropathology. The study of the material afforded by the cases of injuries to the peripheral nerves has been especially fruitful. The research in the neurologic departments of the various armies has demonstrated that anatomic and physiologic ideas current prior to the war were inadequate to explain many of the phenomena associated with injuries to the peripheral nerves.

Compared with the neurologists of the other armies, those of the American army were afforded but a meager opportunity for the study of neuropathology. The few months in which we were actively engaged in warfare, with the necessity for rapid and efficient medical function, did not permit the fullest organization of neurologic units for consistent research of injuries to the nervous system in the cases observed in France. On their return to the United States, the patients with peripheral nerve injuries were assembled in a number of special hospitals where they were closely studied and, when necessary, treated surgically.

The attempts to establish syndromes to differentiate cases of anatomic interruption, compression, irritation, dissociation and recovery, chiefly by M. and Mme. Déjerine and M. Mouzon,[1] have not resulted in any definite conclusions. The American school agrees with many of the French workers that, clinically, anatomic and physiologic interruptions are indistinguishable. Valuable indications of severe lesions have, however, been established (Meige,[2] Pittres,[3] Athanassio-Benisty [4]).

Research in the fields of motion and sensation has explained certain heretofore confusing observations. Supplementary motility was recognized by Swan in 1834. Much of the knowledge concerning this type of movement, which is often responsible for a failure of recognition of the severity of a lesion, was furnished by Duchenne, Beevor and Sherren. Since the war, the importance and common occurrence of these movements has been emphasized by Claude,[5] Athanassio-Benisty, and in this country by Wood,[6] Ingham and Arnett[7] and Pollock.[8]

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[1] Déjerine, J. J.: Déjerine, Mme., and Mouzon, J. Presse méd., May 10, July 8 and Aug. 30, 1915.

[2] Meige: Rev. Neurol. **23,** April-May, 1916.

[3] Pitres: Rev. Neurol. **23**:477 (April) 1916.

[4] Athanassio-Benisty: Clinical Forms of Nerve Lesions and the Treatment and Repair of Nerve Lesions, Military Medical Manual, London, 1918.

In the field of sensation, it is notable in the work of the Allies that, whatever their observations may have been, there is a tendency to concur with the theories of Head. Contributions to sensory studies have been of two types. First, such as detail the sensory distribution of peripheral nerves, notable among which is the work of Stopford,[9] and in this country, Hamilton[10] and Grimberg;[11] second, investigation of the possible existence of separate fibers for epicritic and protopathic sensibilities. These have been studied chiefly in the United States. The problem has been attacked from two angles; first, by Cobb,[12] who has shown, by employing painful stimuli with low degrees of pressure of known quantity, that the areas of tactile anesthesia and analgesia are coextensive; second, by Pollock,[13] whose work tends to show that the early and dissociated return of pain sensibility to higher degrees of pressure is not due to an unphysiologically rapid regeneration of protopathic fibers, but to assumption of function of overlapping nerves. A sign of some confirmatory diagnostic value (Tinel[14] has been added.

In common with other observers, the American neurologists have found electrical reactions to be disappointing as absolute indications of the severity of the lesion. Attention has been directed by the English school to the practical employment of measured shocks from a condenser to determine the degree of injury and regeneration. The importance of coexistence of vascular and joint lesions has been emphasized. The French school has called attention to the occurrence of physiopathic disorders which require differentiation from injuries of the peripheral nerves. Only a negligible number of these so-called

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5. Claude: Rev. Neurol., April-May, 1916.
6. Wood: Arch. Neurol. & Psychiat. **2**:532 (Nov.) 1919.
7. Ingham and Arnett: Arch. Neurol. & Psychiat. **3**:107 (Feb.) 1920.
8. Pollock: Arch. Neurol. & Psychiat. **2**:518 (Nov.) 1919.
9. Stopford: Anatomy, October, 1918.
10. Hamilton: Arch. Neurol. & Psychiat. **3**:277 (March) 1920.
11. Grimberg: Arch. Neurol. & Psychiat. **2**:338 (Sept.) 1919.
12. Cobb: Arch. Neurol. & Psychiat. **2**:505 (Nov.) 1919.
13. Pollock: Arch. Neurol. & Psychiat. **2**:667 (Dec.) 1919.
14. Tinel: Presse méd. **23**:835 (Oct. 7) 1915.

"congealed hands" have been observed by the American neurologists, who agree with Roussy and Lhermitte [15] that they are functional in character as opposed to the view of Babinski.[16]

One of the most important investigations has been directed to the study of intraneural localization or peripheral nerve topography. The specificity of localization of nerve fasciculi was first emphasized by Stoffel. During the war, Marie, Meige and Gosset [17] were able to confirm this work, with certain modifications, by intraneural electrization. M. and Mme. Déjerine,[18] studying partial lesions the locations of which were confirmed by operation, obtained charts of intraneural localizations of a character somewhat varied from the preceding investigators. In this country a notable contribution to this subject appearing in the September issue of the ARCHIVES OF NEUROLOGY has been made by Kraus and Ingham.[19] Their investigations were a continuation of the work of Marie. They point out that one fact is brought out with certainty, the course of motor fasciculi is a straight one from the point where the nerve has been made up by its contributing segments to the point of offset of the fasciculi as a branch. This work permits conclusions opposed to those of Dustin,[20] who called attention to the presence of intraneural plexuses between each branch of a nerve, and of Langley and Hashimoto.[21]

Our knowledge of experimental regeneration of nerves has been enriched by the American investigators, Lewis and Kirk [22] and Huber.[23] It is definitely established that regeneration of a severed nerve occurs from the outgrowth of the axons of the proximal segment. The work of Huber on direct suture and various forms of transplants, fresh and preserved, has indicated the proper surgical treatment of various peripheral nerve lesions. Lateral implantation and nerve plastics have been discarded. The surgical treatment of these injuries seems to offer a better prognosis in the hands of American than other operators. Valuable contributions on surgical treatment have already

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15. Roussy and Lhermitte: Military Medical Manual, The Psychoneuroses of War. Author: Please give publisher and year.

16. Babinski: Rev. Neurol., April-May, 1916.

17. Marie, Meige and Gosset: Compt. rend. Acade. d. méd., Dec. 28, 1915.

18. Déjerine, J. J., and Déjerine, Mme.: Presse méd., No. 13 (March 2) 1916.

19. Kraus, W. M., and Ingham, S. D.: Electrical Stimulation of Peripheral Nerves Exposed at Operation, J. A. M. A. **74**:586 (Feb. 28) 1920.

20. Dustin: L'Ambulance de L'Ocean, 1917.

21. Langley and Hashimoto: Quoted by Kraus and Ingham.

22. Lewis and Kirk: Bull. Johns Hopkins Hosp., February, 1917, p. 71.

23. Huber: Arch. Neurol. & Psychiat., 1919. Author: Please give volume and page.

appeared by Stookey,[24] Dean Lewis,[25] Frazier,[26] Spear and Babcock,[27] Price Feiss and Terhune [28] and Elsberg.[29] Although sufficient time has not elapsed for definite conclusions to be drawn, a promising outlook is afforded by their reports. L. J. P.

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[24] Stookey: Surg., Gynec. & Obstet. **23**:639 (Dec.) 1916.

[25] Lewis: Surg., Gynec. & Obstet. **30**:528 (May) 1920.

[26] Frazier: Ann. Surg. **71**:1 (Jan.) 1920.

[27] Spear and Babcock: Arch. Neurol. & Psychiat. **2**:253 (Sept.) 1919.

[28] Price, Feiss and Terhune: Arch. Neurol. & Psychiat. **1**:547 (May) 1919.

[29] Elsberg, C. A.: Technic of Nerve Suture and Nerve Grafting, J. A. M. A., **73**:1422 (Nov. 8) 1919.

Abstracts from Current Literature

PHYSIOLOGISCH-ANATOMISCHE UNTERSUCHUNGEN UEBER DIE SOGENANNTE ARMREGION DER GROSSHIRNRINDE (PHYSIOLOGIC-ANATOMIC INVESTIGATIONS OF THE SO-CALLED ARM REGION OF THE CORTEX). HANS MEIER-MUELLER, Schweiz. Arch. f. Neurol. u. Psychiat. 5:270, 1919.

In this article Meier-Mueller reports the grosser pathologic and histopathologic findings of the brain of a chimpanzee (*Troglodytes niger*), "Billy," who was operated on by Graham Brown and Sherrington. They removed the arm region of both sides in six successive operations, three on the left and three on the right, in the course of about sixteen months. The arm centers had been located by electrical stimulation at the time of each operation; following the operation there was in every instance a total paralysis of the opposite arm, which recovered with very "little change" in movements ultimately. The animal died nine days after the sixth operation.

A preliminary report of these investigations was made by Graham Brown and Sherrington at the Ninth International Congress of Physiologists in Groeningen in September, 1913. Monakow, who was present at this time, noted that definite synkinesias were present in the movements of the arms, although the animal was able to perform such movements as pouring water into a glass and drinking it. The brain was sent to Monakow's institute, where its examination was undertaken.

The fact that no permanent paralysis could be produced necessitated revision in our views concerning the function of the so-called motor area of the cortex. Grossly, the defect produced by the operations on the right hemisphere included destruction of the subcortical portion of the second frontal convolution, from its middle portion, in an anteroposterior direction and extending upward to the sulcus of the first frontal gyrus, backward through the field of operation, and including the corresponding portion of the postcentral gyrus; the flocculus, centroparietal fasciculus and deeper structures of the brain were not involved. On the left side the defect extended in a fronto-occipital direction from the middle third of the first frontal convolution, including its mesial edge, through the posterior two thirds of the second frontal, in which half the cortex of the sulci remained intact, through the so-called arm center of the anterior central gyrus, into a small portion of the postcentral gyrus lying opposite. The defect, therefore, on both sides included those portions of the arm centers which could be excited electrically, located in the middle third of the precentral gyrus, the caudal third of the second frontal, and a portion of the postcentral, particularly on the right, including the greater portion of the corresponding medullary substance, without involvement however of the centroparietal fasciculus or the pyramidal radiations. The neighboring "leg center" and "head center" were not involved. Examination of the thalamic region revealed an extensive degeneration of the middle segment of the optic thalamus in a fronto-occipital direction, slight changes in the caudal portion of the frontal third, as well as in the dorsolateral portion of the lateral nucleus; the medial portion of the nucleus ventralis b. was more or less extensively involved.

While the thalamic representation of the cortex is now fairly well understood, this furnished an exceptional opportunity of delimiting more sharply that portion of the thalamus corresponding to the arm region. It was noted, in a general way, that on both sides degenerative changes occurred in the thalamus in the caudal portion of the frontal third and in the entire middle third in the anterocaudal direction, in the lateral portion, of the middle third, in a dorsoventral direction; on the right side, there was in addition degeneration of the medial portion of the nucleus ventralis b. The arm region, therefore, would roughly correspond to the middle third of the lateral nucleus of the thalamus. The portion of the postcentral gyrus, destroyed only on the right, would correspond to the medial third of the nucleus ventralis b.

In animals operated by Munk, in which the precentral gyrus had been completely removed, the entire lateral nucleus, in a dorsoventral direction, was degenerated. The writer infers that the cortical representation of the frontal third of the thalamus must be the frontal convolutions. Monakow is of the opinion that the tuberculum anterius corresponds to the medial portion of the first frontal convolution and the paracentral area.

From the physiologic standpoint, movement comprises exceedingly varied and complex factors. Monakow noted certain synkinesias in the arm movement of this monkey. Consequently some of the isolated synergistic movements must have been hampered. This observation appears to refute the generally accepted view that the so-called arm region alone contains the anatomic and dynamic substratum of the manifested "kinetic melodies." Only certain types of movements requiring a fine reciprocal play between agonists and antagonists, or, as Monakow puts it, the ability for reciprocal inhibition of the extraneous and interfering movements of the spinal and subcortical synergies arising in a reflex manner, call for the integrity of the "foci aggregates" of the arm region.

The author next cites three clinical cases which he thinks support this assumption:

The first case was that of a man 21 years of age, whose head had been injured by a wagon wheel three years previously, following which he developed jacksonian attacks of the right arm, which became paralyzed. At operation an encephalitic area was completely extirpated. Following operation, the patient suffered a right hemiplegia, an aphasia and a psychic disturbance, characterized by dulness and disorientation. His condition improved, however, so that eight years later he was graduated from school with an excellent record. The only evident symptom which persisted was a fine tremor in the right upper extremity. Isolated movements of the fingers were equally good in both hands.

The second case was that of a 35 year old farmer's wife, who had jacksonian attacks of the right arm. At operation an oval tumor about 3 cm. long was excised. The right arm was completely paralyzed. Four months later the patient was able to perform isolated movements of the fingers, which, however, were somewhat slow and awkward.

The third case was that of a woman 40 years of age suffering from jacksonian seizures of the right arm, an accompanying right-sided hemiparesis, projectile vomiting and choked disk. An encapsulated abscess was removed from the left precentral gyrus, following which there was a complete right-sided hemiplegia with aphasia. One month later the patient was able to use her hand in dressing and in her housework—however, with a little weakness, slowness and awkwardness.

Every movement is composed, philogenetically, of old and recent, as well as static-dynamic and orientation components. Elementary movements occur in the fetus; these develop into the locomotor reflexes, the rhythmic, orienting, purposeful and expressive movements of the adult. Their localization must necessarily be complex and not delimitable to a given point. It is quite certain that the division of labor necessitates numerous "centers" for given functions, some of which remain subcortical, while others wander to the cortex and toward the frontal area, depending on the philogenetic position of the animal. Just as movement becomes progressively better developed from earliest childhood, so the anatomic localization becomes correspondingly more complicated and diffuse, and doubtless comprises, anatomically, centers that cannot be excited electrically. The author feels that the designation "center" should be replaced by that of "region."

Woltman, Rochester, Minn.

REVIEW OF EIGHT YEARS' EXPERIENCE WITH BRAIN TUMORS.

Ernest Sachs, Arch. Surg. **1**:74 (July) 1920.

Sachs, in this article, gives some of the results of his experience in operating on a series of eighty-five cases of brain tumor. His experience is valuable because he is devoting his entire time to neurologic surgery, one of the very few to do so in this country. As he states, Cushing is quite right in saying that the time has passed when there is any purpose in speaking of the mortality in brain tumor cases any more than there is in speaking of the mortality in abdominal tumor cases. Too often we find pessimism even in the ranks of neurologists. The mortality is admittedly higher than in other fields of surgery, but often operation offers the only chance of relief. Sachs is frank in stating his own mortality—35.5 per cent.; but, as he points out, we find the predominating mortality—eighteen out of twenty-nine—in glioma cases, in which type of tumor we have been accustomed to think of the growth as usually inoperable from the standpoint of removal. Sachs successfully removed 14 per cent. of the gliomas in a series of twenty-one cases; a creditable record. He points out that those gliomas that grow by replacing tissue do not give rise to any general symptoms of increased pressure and we can therefore only depend on local phenomena irritative or paralytic in character. It serves a useful purpose to call attention to the fact that such phenomena may be caused by a tumor which, if operated early enough, may be removable. Sachs states that in the cases of glioma successfully removed symptoms were only present for four months or less and that four weeks should be the longest time to try palliative measures. There is little danger in an exploratory craniotomy in the hands of one trained in neurologic surgery and there is no doubt that such surgeons should be called on oftener to do these operations; that, in fact, such a procedure should be the rule when a brain tumor is suspected and the symptoms have not abated after a short trial of palliative procedures. The reviewer has in mind, of course, only those cases in which surgical removal offers a possible chance of cure and admits that a large number of tumors are so situated as to make surgical removal impossible. Even in such cases decompression may offer great relief.

Sachs calls attention to the value of ventricular puncture on the opposite side from the tumor as well as a preliminary decompression to relieve intradural pressure. There were sixty-four cases presenting tumors other than true brain tumors. In this group there were eleven deaths, a mortality of 17 per cent. Twelve of the cases were cysts. In thirty of the sixty-four cases there were no localizing signs and a decompression was performed to relieve general pressure symptoms. Sachs states that palliative procedures often afford a surprising amount of relief and for a considerable time, a fact with which the reviewer has been impressed. Although the number of cases reported in this paper is not large when viewed from the standpoint of a statistical study, the group is sufficiently large to give results encouraging to their reporter and to those of us interested in neurologic surgery.

Rodman, Philadelphia.

ESSAI D'APPLICATION DES TECHNIQUES DE LA PSYCHO-PHYSIOLOGIE ET DE LA PSYCHOLOGIE EXPERIMENTALE A LA NEURO-PSYCHIATRIE (AN ATTEMPT TO APPLY THE TECHNIC OF PSYCHOPHYSIOLOGY AND EXPERIMENTAL PSYCHOLOGY TO NEUROPSYCHIATRY). Raoul Mourgue, Schweiz. Arch. f. Neurol. u. Psychiat. **5**:240, 1919.

The first part of this study on "The Psychomotor Function of Inhibition Studied in a Case of Huntington's Chorea," was abstracted in the Archives of Neurology and Psychiatry **3**:663 (June) 1920. The author continued the study by investigating the reaction time of the patient to sensory excitations. He had already reported the study of simple reactions, auditory and visual, and of the time of reaction of choice with inhibition. Many of these experiments were conducted under great difficulty, since those ordinarily in use are applied to persons having normal attention and presuppose a certain degree of integrity of intellectual autoconduction. This is far from being the case with the subject of this article, and the precision of these experiments is apparent rather than real.

In the next experiment the patient, having his eyes bandaged, was asked to respond to the blow of a hammer on the table following the command of "attention." The same experiment had been carried on in August, when it was noted that the mean time of response was 0.25 of a second, that for the normal, being, according to Warren, from 0.12 to 0.23 of a second for auditory sensations. One month later, under exactly the same conditions, the reaction time was extended to 0.36 of a second, which is quite different from that first noted. The inconstancy of the various figures obtained is interpreted by the writer as an indication of pathologic distraction.

The time of reaction for simple visual stimuli, employing Pluecker's tube, giving a red light, was 0.71 of a second, which represents a very much higher figure than Warren gives for normal, which is 0.17 to 0.29 of a second.

In testing the time of reaction with choice Mourgue used the modification of Tolouse. It consists of a double handle of copper, which the subject is commanded to move from within out, sometimes with the right hand, at other times with the left, depending on the intensity of the stimulus noted, for the purpose of arresting the hand of a clock, which was set in motion synchronously with the giving of the stimulus. A mean of 1.29 seconds was obtained. The time of discrimination was, therefore, considerably extended—however, discrimination was not abolished since the patient made but two mistakes, one of which he corrected later.

The time of visual reaction with complex choice was obtained by asking the patient to move the copper handle to the right with the right hand on seeing a red light, to the left with the left hand on seeing a white light, and to desist from movement entirely on seeing a green light. Errors in this test were frequent.

A test of perception was performed by asking the patient to name twenty-four objects with which he was familiar; three of these were named inaccurately and one, which was a piece of chalk, was not recognized at all. The time for doing this was 2 minutes and 40 seconds, while the normal time is given as 20 to 25 seconds.

Marie and Lhermitte look on chronic chorea as a disease of both constitutional and acquired origin; constitutional in so far as it represents a congenital lack of resistance of the brain, and acquired through endogenous or exogenous intoxication, which starts off the degenerative process in the corticostriate region. The metabolism of these subjects has been little studied. Pighini and Juan Alzina y Melis have found that there is increased production of creatin and lactic acid, bringing about an increased creatinin and ammonia output in the urine, which, as they remarked, is probably a phenomenon secondary to the abnormal activity of the neuromuscular system. Probably the same explanation attaches to the absence of lipocholesterol inclusions noted in the suprarenals. In view of the paucity of these investigations, the writer feels that his finding of an increased albumin and lymphocytic content of the spinal fluid, indicating a chronic meningo-encephalitis, is of great interest.

Clinical observation of this patient leads the writer to interpret the state of dementia, according to the classic idea, as essentially one of confusion. Synthesis of the psychologic phenomena, in the presence of motor disturbances, demands further attention for, as Maudsley says, "who cannot govern his muscles, cannot govern his attention." Intellectual enfeeblement, which is not accompanied by distorted ideas, demands a minute and methodic investigation heretofore unpracticed.

The motor theory of psychic phenomena gives a satisfactory explanation of the intellectual and affective phenomena (loss of attention and irritability) here noted, which long have attracted the attention of clinicians. It alone permits a correlation of the anatomic and pathologic findings of Huntington's chorea, as described by Pierre Marie and Lhermitte, of the clinical observations, and of the findings obtained by psychologic experimental methods. The function of psychomotor inhibition seems, from a morphologic point of view, to be related to the neokinetic system of Ramsey Hunt. This is a rational application of the theory of "chronologic localization," expounded by Monakow, on which depends the future neuropsychiatry.

WOLTMAN, Rochester, Minn.

PRINCIPLES OF PERIPHERAL NERVE SURGERY. DEAN LEWIS, J. A. M. A. **75**:73 (July 10) 1920.

The author reviews briefly the principles involved in the process of degeneration and regeneration of peripheral nerves, emphasizing conditions that hinder and assist regeneration following neurolysis or nerve anastomosis. Scar tissue forms rapidly after the division of a peripheral nerve, and the presence of a neuroma may be expected within nineteen days. Since the neuraxes must enter the distal segment in the process of regeneration, a neuroma naturally interferes with its progress and should be resected to a point at which healthy funiculi appear and no scar tissue is visible to the naked eye. It is important to preserve the nerve pattern in the anastomosis, which should be performed as early as possible after the injury, about three and one-half months, but not until all evidence of infection has disappeared. Results at this time will be better than can be obtained six months later. The author states emphatically that "end-to-end suture is the only procedure that can be relied on to reestablish the continuity and function of a nerve after division." It is immaterial whether silk or catgut is used in the anastomosis, and a covering is not necessary. Autogenous transplants and fascial tubulization prove successful in dogs, but not in man.

One hundred and twenty operations are reported; in twelve of these autocable transplants were used and in six tubulization with fascia. In none has there been recovery, although in two transplantations there has been a slight return of motor power in isolated muscles. Tinel has stated that neurolysis is permissible only when it restores a free, supple nerve in the interior of which is found no obstacle to regeneration and that, as a whole, liberation is ineffective in cases of severe lesion of nerve trunks associated with rupture of the sheath, and the development of scar tissue with the formation of a neuroma. Neurolysis is of distinct value, but it is sometimes difficult to determine whether or not the results are the direct results of surgical intervention; nerves presenting the most marked pathologic changes have recovered spontaneously.

Whether neurolysis or resection is to be performed depends on the appearance and feel of the nerve and the electrical reaction. It should be combined with capsulectomy, that is, removal of the thickened epineurium, or parallel incision to it, and transference of the nerve between normal muscle fibers away from the bed of scar tissue. The musculocutaneous nerve is the first to regenerate, then the musculospiral, the internal popliteal, and the median. The external popliteal is the slowest to recover.

Aside from the operative procedure, the author emphasizes the importance of postoperative care, the use of massage, and measures to prevent overstretching of muscles; he also calls attention to the fact that fibrous changes in a muscle or ankylosed joint defeat the object of peripheral nerve repair.

The article is especially valuable to the clinician in that it states briefly the process of degeneration and regeneration, emphasizes the surgical indications, and shows the comparative value of end-to-end anastomosis with that of fascia tubulization and autogenous transplants.

Adson, Rochester, Minn.

LE MONGOLISME (MONGOLIANISM). E. Apert, Bull. méd. **34**:563 (June 19) 1920.

In the extremely varied group of abnormal infants, the author considers congenital myxedema and mongolianism as two special groups, each different from idiocy, imbecility, retardation of evolution or mental anomaly based on congenital lesions or on defects of cerebral development. His paper deals with the second of these groups. A large portion dealing with the clinical features may be passed over as familiar. Regarding teeth in these cases: The eruption of teeth is delayed. The first teeth sometimes do not come until the tenth, fifteenth, or twentieth month. The order of their appearance is irregular and quite often certain teeth are lacking, particularly the lateral incisors. The molars appear late, being delayed until the third, fourth or even fifth year. The second dentition is retarded and irregular, but one does not find in general, the microdontism, the dysmorphisms, or erosions of enamel seen in hereditary syphilis. The dental fragility and early caries seen in myxedema are also absent.

In the great majority of cases seen by the writer he has been able to exclude syphilis as an etiology. He has found that the mongolian is frequently the last of a numerous family, the mother relatively advanced in age and weakened by frequent pregnancies. Often he has found that in the months of pregnancy mental and moral factors, distressing to the mother, were present.

He believes that he can benefit cases of mongolianism by opotherapy, using thyroid and suprarenal glands. He writes: "I use with the powdered organs manganese, which appears to be the metal-ferment of the suprarenal gland, and chalk and magnesium, which appear to be the metal-ferments of thyroid and parathyroid." For an infant in the first year he gives at the beginning:

	Cg.
Suprarenal (whole gland), dried and powdered	0.02
Thyroid, dried and powdered	0.01
Bioxid of manganese	0.01
Glycerophosphate of chalk	0.05
Glycerophosphate of magnesium	0.05
Sugar, powdered	0.30

This is used for four days continuously and then omitted for three days. The dosage is gradually increased every second week or so, and even as much as 10 cg. of suprarenal gland by day can be given an infant of 1 year.

After outlining the features of improvement that may be expected, he writes, "the mongolians will always need guidance. They will lack initiation and judgment. . . . They will remain always below standard but can be sufficiently restored so as to escape the special asylums where until recently they were confounded with the true idiots."

Davis, New York.

FINGER EXPLORATION OF GUNSHOT WOUNDS OF THE BRAIN.

Edward B. Towne and T. R. Goethals, Ann. Surg. **71**:531 (May) 1920.

The authors emphasize the value of finger palpation of gunshot wounds of the brain and state that foreign bodies are frequently overlooked by both curettage and catheter palpation. They report a series of sixty-one of the head wounds operated on by the surgical team at the casualty clearing stations of the British First Army from May 12 to Aug. 12, 1918. Eleven of the wounds were simple scalp wounds, and twenty-two were fractures with unopened dura; none of these thirty-three cases brought out data of particular interest. Their discussion is confined to twenty-eight cases of skull fracture associated with lacerations of the dura and brain. Twenty patients were operated on; eight were inoperable. The cases are reported in detail, and attention is called to the fact that thirteen of the patients evacuated from the clearing station were followed for periods of from six months to twenty-one months after operation.

The authors give an excellent review of the literature on the treatment of various types of skull injuries. They outline the advantages and disadvantages of the four principal methods employed in the treatment of severe brain wounds, namely, (*a*) extraction of metal with the magnet or under the screen, (*b*) gentle curettage of the cavity, (*c*) catheter palpation with forceps extraction, and (*d*) finger palpation with forceps removal of the foreign body. The method best adapted to the individual case should be followed, bearing in mind the fact that finger palpation is indispensible when the wound is sufficiently large to admit the finger. Since the brain wound frequently is much larger than the dural opening, the dural wound should, if necessary, be opened.

The routine treatment instituted by the authors was briefly as follows: Unless obviously a subject for general anesthesia, local anesthesia of procain was employed. The damaged scalp and pericranium were removed, the wound treated with tincture of iodin, and the involved skull removed in block. A 17 F. soft rubber catheter with an attached syringe was used to aspirate the necrotic cavity and, if the defect in the dura was large enough, a finger was introduced gently and the foreign bodies removed with forceps. If the defect in the dura was too small to admit a finger but information pointed to a cavity in the brain large enough for finger exploration, the dura was incised to allow the finger to enter. The wound was irrigated with saline, hemorrhages were controlled, and the dura was sutured partially or, if possible, completely with fine chromicized catgut or silk. A few plain catgut stitches were used in the galea and silkworm gut in the skin. A small gutta percha tissue wick was introduced to the bone if hemostasis was doubtful. During the convalescent period, the wound was inspected daily; if there was any evidence of tension or presence of headache, a spinal puncture was made and repeated as often as indicated. The sutures and wick were usually removed in from twenty-four to forty-eight hours.

The article is timely in that it reviews extensively the literature on the treatment of skull and brain wounds and emphasizes practical surgical procedures.

Adson, Minnesota.

LE SYNDROME PARKINSONIEN DANS L'ENCEPHALITIS LETHARGIQUE (THE PARKINSON SYNDROME IN EPIDEMIC ENCEPHALITIS). Pierre Marie in collaboration with G. Levy, Bull. de l'Acad. de méd. **83**:539 (June 15) 1920.

The writer concerns himself with points of difference between the postencephalitic parkinsonian syndrome and paralysis agitans (true Parkinson's disease).

In the beginning he points out that the lesion of paralysis agitans is considered by some to be the central nuclei, particularly the lenticular nucleus, while Tretiakoff believes the lesion is in the cerebral peduncles, especially in the region of the locus niger.

The age at onset is outstandingly unlike, generally speaking. Paralysis agitans rarely begins before 45. The parkinsonian syndrome following epidemic encephalitis occurs most often between the ages of 20 and 40. He has seen a case of epidemic encephalitis showing the parkinsonian syndrome in a child of 12.

Secondly, the preexistence of a general affection, usually febrile, can be found in all the instances of the postencephalitic condition. Headache, vertigo, diverse pains more or less localized, and ocular troubles are features not found in paralysis agitans.

The mode of onset is very different. In paralysis agitans the phenomena progress slowly. The other member on the same side of the body also becomes involved several months following the involvement of the initial extremity. Because of this sequence, the writer remarks that in paralysis agitans the mode of onset is "progressively hemiplegic." In sharp contract to all this, the onset of the parkinsonian syndrome in encephalitis is abrupt and also the extremities as well as the trunk are involved en masse, at the same time. The progressive hemiplegic mode is altogether lacking.

Fourthly, the writer has yet to find a postencephalitic case showing the tremor which is typical of paralysis agitans—the minute tremor which is temporarily in abeyance at the moment of voluntary movement. In postencephalitic cases the tremors are of large amplitude and are either released or augmented by voluntary movement. Spasmodic jerking movements of the face are seen in postencephalitic cases and not in paralysis agitans.

Abnormalities in the use of the tongue, in the opening and closing of the mouth and in mastication, present in some of the postencephalitic cases, are absent in paralysis agitans. That these particular differences arise is not surprising in view of the frequency in epidemic encephalitis of lesions of the peduncular-bulbar trunk.

Lastly, there is the difference of evolution. In the postencephalitic condition the syndrome has often almost completely cleared up, while in true paralysis agitans neither a cure nor even a recession of the symptoms is possible.

Davis, New York.

HAND AND FOOT PRINTS AS RECORDS OF THE PERIPHERAL NERVES. Lewis J. Pollock, J. A. M. A. **74**:943 (April 3) 1920.

Graphic methods of recording signs and symptoms have in many instances a greater value than descriptive methods. It is frequently impossible to get photographic records of hands and feet in cases of peripheral nerve lesions; it is then serviceable to record the contour of the palm and that of the sole by the aid of impressions. Imprints show distinctive changes in lesions of five of the peripheral nerves, namely, the ulnar, the median, the radial, the internal popliteal and the sciatic, and also in combined lesions of the ulnar and the median.

Imprints of the hand in cases of ulnar nerve lesions show clawing of the inner two fingers, atrophy of the hypothenar muscles, nonseparation of fingers and extension of the first phalanx of the thumb. Atrophy of the adductor pollicis is demonstrable from the imprint.

Median nerve lesions show clearly the disturbance of the whorl formation on the tips of the index and middle fingers. When severe clawing is present in these two fingers, it is made evident by the absence of any imprint of the central portion of the palm. The failure of desquamation and the presence of many new lines over the thenar eminence are demonstrated.

Imprints of radial nerve lesions are characterized by the cramped appearance of the fingers which results from the inability to place the palm in a flattened position on the paper. The most characteristic feature of this imprint is the position of the thumb, which is adducted, the distal phalanx falling within or on the border of the outline of the index finger. The thumb is rotated about its own axis inwardly; and its distal phalanx is usually flexed. Absence of the signs of atrophy of the thenar and hypothenar eminences is an additional feature of this form of lesion.

In combined lesions of the ulnar and median nerves, signs of atrophy of both the thenar and the hypothenar eminences are demonstrable in the impressions by means of the notches found along their borders. Clawing is present in all four fingers.

Lesions of the sciatic nerve show, besides a slight pes cavus in some cases, a clawing of the toes indicated by the absence of their imprint on the paper. It may be stated that, although by no means diagnostic, such records are of considerable value in determining the progress of the condition of atrophy and deformity in peripheral nerve lesions. The imprints are easy to take and require a minimum of time for the amount of record produced.

OTT, Rochester.

REPORT OF THE CONDITION OF A CASE OF COMPLETE DIVISION OF THE SPINAL CORD BY GUNSHOT WOUND NINETEEN YEARS AFTER THE INJURY. WILLIAM B. CADWALADER, Ann. Surg. **71**:719 (June) 1920.

The author describes a case which was reported by Drs. Francis T. Stewart and Richard H. Harte in the *Philadelphia Medical Journal* of June 7, 1902, under the title of "A Case of Severed Spinal Cord in which Myelorrhaphy was followed by Partial Return of Function."

The patient in question was shot in the back with a 32 caliber pistol, the bullet entering 1 inch to the right of the seventh dorsal spine and passing directly into the spinal canal. There was immediate and complete motor and sensory paralysis below the level of the tenth dorsal spine, and all the superficial and deep reflexes were abolished. Three hours after the injury an operation was performed by Stewart. The laminae of the seventh and eighth dorsal vertebrae were removed; the membranes were found to be lacerated, and the spinal cord was completely severed. There was a distance of three-fourths inch between the severed ends of the cord. The divided ends of the spinal cord were approximated with catgut sutures and the wound closed. Surgical recovery was uneventful.

The patient was examined repeatedly by different physicians and was exhibited before many medical societies with the impression that there was some return of function. In making an examination recently, the author reports a complete paralysis of motion and sensation below the level of the tenth thoracic spine. The lower limbs feel rigid, but there is no wasting. The skin of the legs is somewhat waxy in appearance and pits slightly on pressure. The defense reflexes have developed, with the development of an automatic control of the bladder. The author states that the occurrence of any return of function must emphatically be denied; that such a statement was purely the result of error in making the observations.

The article is especially valuable in that it emphasizes the importance of a thorough neurologic examination, and substantiates the fact that regeneration cannot occur following myelorrhaphy. It is possible to occur only when the lesion is in the cauda equina, and one is able to anastomose the nerve roots after they have emerged from the cord substance.

ADSON, Minnesota.

ON THE RELATIVE VASCULARITY OF VARIOUS PARTS OF THE CENTRAL NERVOUS SYSTEM OF THE ALBINO RAT. EDWARD HORNE CRAIGIE, Jour. Comp. Neur. **31**:429 (June 15) 1920.

The author has made the first exact quantitative comparison of the vascularity of different parts of the central nervous system. Measurements of the richness of capillary supply were made in twenty-one regions of the spinal cord, medulla oblongata and cerebellum, and the results are presented in the form of tables and graphs.

The gray matter is more richly vascularized than the white matter, the poorest part of the gray being nearly half as rich again as the richest part of the white. All parts of the white matter are not equally vascular, the pyramidal tract, the richest part in the spinal cord, being about twice as rich as the fasciculus cuneatus, while the fasciculus longitudinalis dorsalis in the medulla is still richer.

The gray centers can be sharply divided into two groups, the motor nuclei and the sensory and correlation centers, of which the latter are richer than the former. Though the richest motor region (ventral cornu) is but little poorer than the poorest sensory one (the fifth spinal nucleus), the two groups do not overlap in the case of those regions studied, except in a few individuals. The substantia gelatinosa Rolandi of the spinal cord is the only part which does not conform with this statement. The richest of the centers observed is the dorsal cochlear nucleus, which is more than half as rich again as the ventral cornu, about two and one-half times as rich as the substantia gelatinosa Rolandi (the poorest gray region), and eight times as rich as the fasciculus cuneatus. Great individual variations occur, and the two sexes do not seem to show any constant difference.

The interpretation of the functional significance of the anatomic data recorded depends on the unsolved problems of the fundamental nature of nervous processes and their relation to metabolic activity. The bearing of various theories regarding these on the present investigation is discussed.

Herrick, Chicago.

RECOVERY FROM TUBERCULOUS MENINGITIS AFTER TREATMENT WITH INTRASPINAL INJECTIONS OF ANTIMENINGOCOCCIC SERUM. A. W. Hollis and T. H. Pardee, Arch. Int. Med. **26**:49 (July) 1920.

Despite the comparative frequency of tuberculous meningitis, the fact that the literature contains only thirty-eight cases in which there has been recovery shows the high mortality of the disease. The authors have used intraspinal injections of antimeningococcic serum as a new therapy in tuberculous meningitis. This has been used by only one previous worker (S. Schaeffer, 1913) and then in a single case. In their series of seven cases the authors report recovery in four. Of these four, the diagnosis in two cases was confirmed by demonstration of tubercle bacilli in the spinal fluid; in the other two cases convincing clinical proof for the diagnosis is offered. Concise case reports are given.

Removal of the spinal fluid always preceded the injection of serum. The amount of serum injected was 20 c.c. or somewhat less, and the first two or three injections were given at daily intervals and then every second or third day. No patient received more than five injections. The cases with demonstrated tubercle bacilli in the spinal fluid received four and five injections, respectively, and recovered. The development of serum sickness appeared to hasten the therapeutic response.

Concerning the manner of this response, theories are briefly considered. The writers believe that "the intraspinal injection of antimeningococcic serum (in cases of tuberculous meningitis) has two distinct actions; first by adding to the spinal fluid certain antibodies which it is unable to develop itself and second, by the introduction within the dura of a foreign protein in the form of horse serum. The irritative effect of the latter on the meninges produces a cellular response and a hyperemia about the site of any localized tubercle with beneficial result."

This work is eminently important and should be widely noted in the hope that other clinicians may use the method and add to our knowledge of a promising new therapy.

Davis, New York.

DISCUSSION OF A CASE OF PARALYSIS OF BOTH PERINEAL NERVES. S. Slansky, Neurol. Centralbl. **39**: (Feb. 1) 1920.

The author reports a patient, 43 years of age, who was operated on for hernia, Oct. 8, 1918, under chloroform anesthesia. The thighs were strapped together just above the knee. On October 18, the patient experienced difficulty on attempting to walk, due to a double foot drop. November 11, examination revealed a bilateral peroneal paralysis, more severe on the right side. No other pathologic condition could be demonstrated. There was no sensory disturbance, as shown by direct and indirect faradic stimulation, although the contractions of the right leg were more sluggish. By galvanic stimulation no reaction of degeneration was elicited. The deep reflexes were reduced, but there were no pathologic sole reflexes or ataxia.

In his discussion, the author refers to the frequency of brachial plexus paralysis during operation. Brown states that this is accounted for by compression of the brachial plexus between the clavicle and the cervical vertebrae, due to severe abduction and extension of the upper arm. Attention has been given to the prevention of postoperative brachial palsies, but thus far the utmost care has not always proved successful.

In the author's experience this is the first instance of bilateral peroneal palsy occurring during operation and he questions whether or not the chloroform was the contributing factor and what influence the strap may have exerted. He states that moderate pressure over a considerable period might well be assumed as the cause, especially if forcible flexion or extension of the legs is permitted during the operative procedure. In this particular instance the paralysis was completely relieved after three weeks' treatment.

Shelden, Rochester, Minn.

AMPUTATION NEUROMAS: THEIR DEVELOPMENT AND PREVENTION. G. Carl Huber and Dean Lewis, Arch. Surg. **1**:85 (July) 1920.

The Great War has stimulated the study of peripheral nerve surgery enormously. It was the frequent occurrence of painful stumps in war amputations that led the authors to carry on this study in order to try to discover the cause of, and to prevent the development of, amputation neuromas. They state that as infection and hemorrhage are known to play an important rôle in the development of these neuromas that their experimental work was carried out with strict precaution to prevent both. In spite of the fact that they were entirely successful in this amputation neuromas invariably developed after experimental resection of the sciatic nerve in rabbits. Neuromas, as the authors point out, are due to the effort of the neuraxes to join the divided distal end. Being unable to do so the neuraxes are turned back on themselves by the connective tissue. The authors have shown by experimental work that the best way to prevent the development of such neuromas is to inject the nerve with absolute alcohol ¾ to 1 inch proximal to the proposed site of resection; if this is done there is an immediate nerve degeneration; the neuraxes in the injected areas are broken up into short irregular segments, myelin is present in the form of a granular detritus and smaller and larger globules. The neurilemmal sheaths appear well preserved. The longest time that elapsed from injection to removal of the nerve and microscopic studies was 175 days. By this time numerous neuraxes both myelinated and nonmyelinated had passed through the injected part of the nerve to the extreme distal end of the proximal segment. The connective tissue sheath of the nerve is materially thickened at this point. The structural appearances presented are not those of a neuroma. The use of alcoholic injection to cause nerve degeneration is a proved procedure, but its use to prevent the formation of neuromas is new and in the light of this report should be given a clinical trial.

Rodman, Phildelphia.

APOPLEXY AND HYPERTONIA. Erna Herrmann, Neurol Centralbl. **39**: (Feb. 1) 1920.

Strasser recently called attention to the fact that those with hypertension are subject to apoplexy only when the condition is associated with arteriosclerotic changes of the vessels or with chronic nephritis. Of ninety-seven hypertonics with arteriosclerosis, eight had severe apoplexy, and of twenty-nine nephritic cases, six were similarly affected. Thirty-four cases of simple hypertonia showed no severe apoplexy, although some had very high blood pressure.

The author studied forty-four cases of apoplexy to determine the relationship to arteriosclerosis, chronic Bright's disease and hypertonia. She calls attention to the difficulty of accurately distinguishing essential hypertonia from that based on organic disease, but she depends on the physical examination to determine this. The forty-four cases included twenty-two men and twenty-two women. One hundred and forty mm. of mercury were considered the upper limit of normal. Thirty-five of the cases showed a definite arteriosclerosis with symptoms dependent on this condition; three had an old valvular disease; four had chronic nephritis, and two had an old syphilis with positive Wassermann reaction. Of the forty-four cases none were found which could be classified as an essential hypertonia.

Shelden, Rochester, Minn.

CONGENITAL FACIAL PARALYSIS. Frank R. Fry, J. A. M. A. **74**:1699 (June 19) 1920.

The author reports two cases of congenital facial paralysis of unusual interest. The first case, that of a child aged 1 month, was brought to the pediatric service of the outpatient department of Washington University School of Medicine with a complete right and a partial left facial paralysis. The second case was that of a young man, who gave a history of congenital weakness of the right facial muscles. In both instances the paralysis was more marked in the upper branches of the seventh nerve.

In the author's belief, congenital facial paralysis is due to a disease of the nuclei. Peripheral facial paralysis affects the whole side of the face, whereas in the central and in most of the bulbar types the superior branch retains its movement. In congenital cases the superior branch remains motionless, while the region about the mouth retains either complete or partial motility. If this difference between the upper and the lower part of the face is great, it gives a characteristic facial expression, such as full protruding lips, in contrast to the masklike, expressionless, sunken face mentioned above. The appearance of the face makes the condition often recognizable at the time of birth, and gives the impression of an advanced atrophy or aplasia of the muscles; also, the skin over the muscles is usually smooth, peculiarly shiny, and colorless.

Adson, Rochester, Minn.

THE CRANIAL, OCCIPITAL AND ANTERIOR SPINAL NERVES OF THE DOGFISH, SQUALUS ACANTHIAS. H. W. Norris and Sally P. Hughes, J. Comp. Neurol. **31**:293 (June 15) 1920.

The dogfish, as a typical generalized vertebrate, has long been a classic subject for investigation of problems in vertebrate embryology, comparative anatomy and physiology. In recent years it has also been used extensively in medical schools as a dissection type to introduce the student to the general features of the anatomy of the central and peripheral nervous system and sense organs.

Norris and Hughes have given a detailed description of the peripheral nervous system in a paper of 110 pages with fifty-three illustrations. On the basis of serial microscopic sections of the entire head of young specimens, controlled by dissections of the adult, they have analyzed the nerve components, giving detailed and exact information regarding the entire peripheral courses of nerve fibers of all functional systems. Future students will have the benefit of this information regarding the central and peripheral connections of every functionally distinct kind of nerve fibers in the cranial nerve roots, ganglions and peripheral nerves. Comparisons are made throughout with the smooth dogfish, Mustelus.

Herrick, Chicago.

MEDIANUSLAEHMUNG MIT FOLGENDER PHRENICUSLAEHMUNG NACH SCHUSSVERLETZUNG (MEDIAN NERVE PARALYSIS FOLLOWED BY PARALYSIS OF THE PHRENIC AS THE RESULT OF GUNSHOT INJURY). Richard Meisner, Neurol. Centralbl. **39**:84 (Feb. 1) 1920.

On Oct. 29, 1918, a soldier, aged 23, was wounded 7 cm. below the coracoid process in the left arm, the injury producing permanent paralysis of the median nerve. The bullet emerged near the lower end of the left scapula. In January, 1919, there developed painful and restricted breathing and pain in the region of the stomach, especially after meals. Deep breathing and gaping produced pain in the left hand. The tenth, eleventh and twelfth dorsal vertebrae and ribs were painful on pressure. Also, 6 cm. above the middle of the clavicle pain was produced on pressure, which extended down the left arm. The left hand exhibited anesthesia, weakness and atrophy in the distribution of the median, and abnormal perspiration, cyanosis and glossiness. Roentgen-ray examination revealed a paralysis of the left diaphragm in a high and motionless position. This persisted until March, when its function became normal.

Two views are offered to explain the involvement of the phrenic nerve: First, by an extension of inflammation from the wounded area to the phrenic nerve by way of the lymphatics. Second, by an ascending neuritis along the median to the roots which are common to both nerves. The author favors the latter view.

Shelden, Rochester, Minn.

LETHARGIC ENCEPHALITIS IN PREGNANCY. Margaret Schulze, J. A. M. A. **74**:732 (March 13) 1920.

Lethargic encephalitis rarely occurs during pregnancy. Including the case reported by the author, there are only eight cases on record; of these, one patient recovered, five died, and in two instances the outcome was not reported.

The author reports the case of a woman, 35 years of age, one month past term, who died of pulmonary embolism on the twenty-sixth day after delivery. Microscopic examination of the brain revealed characteristic findings of lethargic encephalitis.

This disease appears to be far more common in the male than in the female—122 cases in males and 67 in females were reported.

Ott, Rochester.

THE OLFACTORY SENSE ORGANS OF ORTHOPTERA. N. E. McIndoo, J. Comp. Neurol. **31**:405 (June 15) 1920.

Dr. McIndoo in this and several preceding articles has contributed new observations on the olfactory organs of insects. Contrary to the prevailing belief, the antennae are not the chief organs of smell. This sense is served by "olfactory pores" widely distributed over the body surface. In the Orthoptera the total number of pores varies from 271 to 1,616.

Herrick, Chicago.

LES MODALITES DE DEBUT DE L'ENCEPHALITE EPIDEMIQUE (TYPES OF ONSET IN EPIDEMIC ENCEPHALITIS). J. A. Sicard, Bull de l'Academie de méd. **83**:474 (May 18) 1920.

This brief paper concerns clinical types of epidemic encephalitis and emphasizes the occurrence of forms other than the oculolethargic originally described. For this reason, the writer deprecates the name lethargic encephalitis for the group.

It is in the early stages of the disease that the types stand out most distinctly—the oculo-lethargic, myoclonic, algic and paraplegic. The first type is already thoroughly recognized The myoclonic type often evolves totally free from lethargy or ocular troubles. Rhythmical twitching movements are present instead. These, when diaphragmatic in localization, are pathognomonic. The type with pain as an early feature—the so-called algic modality—can be confounded with many things—facial neuralgia, otitis, tabetic radiculitis, rheumatic neuritis, diaphragmatic pleurisy and renal colic. Diagnosis at the onset of this type is often impossible and must await the appearance of corroborative signs. The paraplegic type is more rare, though the writer has seen two cases. Here again diagnosis must be uncertain until the appearance of ocular troubles, lethargy or special muscular agitations point the way to the true situation.

Davis, New York.

Society Transactions

AMERICAN NEUROLOGICAL ASSOCIATION

Forty-Sixth Annual Meeting, June 1-3, 1920, New York

J. Ramsey Hunt, M.D., *President*

STATIC AND KINETIC SYSTEMS OF MOTILITY. Presidential Address. Dr. J. Ramsey Hunt. This paper appears in full in this issue.

ACQUIRED DOUBLE ATHETOSIS. Dr. William G. Spiller, Philadelphia. This paper appears in full in this issue.

DYSTONIA MUSCULORUM DEFORMANS. Dr. E. W. Taylor, Boston. This paper appears in full in this issue.

BRADYKINETIC ANALYSIS OF SOMATIC MOTOR DISTURBANCES. Dr. S. Philip Goodheart and Dr. Frederick Tilney, New York. This paper will appear in an early issue.

DISCUSSION

Dr. Smith Ely Jelliffe, New York, said that on hearing certain of the remarks concerning these papers he was reminded of some discussions among botanists in his student days between the so-called "splitters" and the so-called "lumpers." One group of botanists would go into the fields and bring back some twenty different species of violets; the other group would take these "species" of violets, place them all under one "species" and call the variants "sub-varieties." One is reminded of this by some of the suggestions made here: Oppenheim maintains that dystonia musculorum is a distinct disease, double athetosis is a distinct disease, dystonia musculorum with or without liver changes is a distinct disease, and Wilson's progressive lenticular degeneration is a distinct disease. All this is from the standpoint of the "splitters." This type of mind acts statically and pigeon-holes things into disease entities. Dr. Jelliffe believed that this entire group might best be regarded as a series of variants with general trends in one or another direction, according to the interruption of precise fiber tracts or synaptic junctions, which are located in or are passing to and from the striatum region. This region is one of extreme complexity, and not as simple as has been regarded by some of the speakers; to speak of the functions of the striatum, as though it were a simple structure, is naive. Of the various recent analyses of the striatum possibilities, which would have to include all of the complex pathways of this region, reference should be made particularly to the work of Cecile and Oskar Vogt,[1] who presented the best representation of the anatomy of this region. At a recent meeting of the New York Neurological Society, Dr. Abrahamson presented three cases of dystonia musculorum, which have been illustrated in Drs. Tilney.

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[1] Vogt: Heidelberger Akad. d. Wissenschaften Biol., Series No. 14, 1919.

and Goodhart's moving picture analysis, and called attention to the opportunity for the study of the developmental phases of the dystonia musculorum group. Particular emphasis should be placed on the variety of clinical trends of a larger group of striatum syndromes of which these cases are merely striking types. It has become increasingly evident that dystonia musculorum, Vogt's double athetosis, Westphal's pseudosclerosis, Wilson's lenticular degeneration, Huntington's chorea, tuberous sclerotic idiocy (not mentioned by Dr. Spiller) and even paralysis agitans, are to be regarded as variants of the larger picture of striatum syndromes. One of Ziehen's patients came to necropsy and negative findings were reported, but in the more recent Flatau and Sterling case studies of the striatum and liver were made by Thomalla,[2] Schneider,[3] and von Economo.[4] This case presented a combination of the Ziehen-Oppenheim group of dystonia musculorum and Wilson's lenticular degeneration, since the clinical picture was characteristic of the former, while the hypertrophic sclerosis of the liver was typical of the latter. Abiotrophic atrophy of the cells of the putamen was found in the striatum. A synthesis of the various striatum syndromes, outlining the pathologic trends and enlarging the neurologic picture, has recently been attempted by the Vogts[5] and Bielschowsky.[6] The former[7] distinguish (1) the *état marbé* originally described by them as double athetosis (in this group Little's disease may be included); (2) *état dysmyélinique,* or progressive degeneration of the fibers passing between the striatum and the pallidum and the thalamus and hypothalamus (in one of Jelgersmas cases a paralysis agitans syndrome was present); (3) the *état fibreux,* progressing often as a bilateral progressive chorea without psychical symptoms; (4) an initially acute, then stationary status fibrosis as a partial expression of cerebral hemiatrophy (Bielschowsky) with nonprogressive athetosis and minimal paresis of body musculature; (5) total necrosis of the striatum confined chiefly to the putamen, at times reaching other parts of the globus pallidus (the dystonia case of Thomalla and Wilson's lenticular degeneration type both belong to this group); (6) acute infectious striatum syndromes (in our recent lethargic encephalitis epidemic many of these paralysis agitans-like cases, athetotic cases, pseudobulbar palsies, involuntary laughing and crying cases, etc., have been observed); (7) presenile degenerations involving chiefly the striatum and pallidum combined with neuroglia proliferations in striatum, and (8) status disintegratus with paralysis agitans-like symptoms, with the striatum involved either as (*a*) a general massive atrophy, chiefly of the caudatus, both ganglion cells and fibers, (*b*) small lacunar softenings or hemorrhages, and (*c*) rarefication and absorption, related to Marie's lacunar softening. The relationships of the striatum to the vegetative control of the muscle metabolism through the liver raises a point of speculative interest. Whereas Wilson argued that a toxin from perverted liver function caused the striatum changes, Dr. Jelliffe emphasized the neural integration disturbance of striatum interruption of vegetative liver pathways. Schneider's study offers several points of interest concerning the possibilities of an analysis of the vegetative integration mechanism essential for liver function, interference with which causes vagatonic gliosis, to the disadvantage of the entire muscular metabolic functions, since it seriously impaired the integration of the involuntary part of the muscle structure, the sarcoplastic substance, and the sensorimotor, anisotropic disk parts. This latter speculative concept might have been raised by Dr. Hunt in his opening address.

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[2] Thomalla: Ztschr. f. d. ges. Neurol. u Psychiat. **41**: 1918.

[3] Schneider: Ztschr. f. d. ges Neurol. u. Psychiat.

[4] Von Economo: Ztschr. f. d. ges. Neurol. u. Psychiat. **43**:173,.1918.

[5] Vogt: J. f. Psychol. u. Neurol. **24**:1, 1918.

[6] Bielschowsky: J. f. Psychol. u. Neurol. **24**:20, 1918.

[7] Vogt: Zur Kenntniss der pathologischen Veränderungen des Striatum, Sitzsb. d. Heidelberg Akad. d. Wiss., 1919.

Dr. Ernest Sachs, St. Louis, said that he had two observations which might have some bearing on the origin of athetosis. Some ten years ago he showed a girl with unilateral athetosis before the New York Neurological Society, and about a year later he removed her cortical arm center. About six weeks ago, he had a similar case. In both these instances the athetosis was absolutely stopped. Dr. Sachs said that he did not know exactly how to interpret this, but Monakow's theory might explain it. Sir Victor Horsley in 1908 published a case in which removal of the arm center for a similar condition also completely stopped the athetosis.

Dr. Frederick Tilney, New York, said that it seemed to him that from the pictures which have been seen, the classical terms for most of the defects due to hyperkinesis were inept and ineffectual for purposes of exact analysis of disturbed motility. If they are to be retained for future usefulness, they should be more fully defined. Chorea, for example, from χορεία, a dance, in itself has very little descriptive value. By connotation, it is associated with a large group of motor disorders that on finer analysis appear to have many fairly distinct characters, such, for example, as the flaccid chorea and the rigid chorea, the single muscle twitch, the muscle group twitch, and diffuse and associated muscle twitch. If chorea is a desirable term for these motor phenomena, then the several varieties must be carefully analyzed and grouped. The same is the case with athetosis, especially honored by American parentage, from the Greek ἄθετος, out of place, which is not descriptive of the motor phenomena indicated by it. Our conception of just what is meant by athetosis is not convincing. The invention of the hybrid choreo-athetosis emphasizes precisely how confused are our notions of this type of motor disorder. Generically, the conditions are probably closely related, but there are certain specific differences which must count heavily in the consideration of exact cerebral localization. Even dystonia musculorum deformans is not altogether free from this criticism, but requires much closer scrutiny and analysis. The danger inherent in all of these terms is that they offer an easy verbal escape from the necessity of careful thinking. The more convenient the terms are, the less apt are they to be exact. Our conceptions and definitions of the defects due to hyperkinesis require closer analysis before they assume real value in understanding the somatic motor organization and its defects. Hyperkinesis may disturb the attitudes and postures of rest by introducing purposeless and adventitious movements, it may deform the performance of volitional acts in the same way, and it may distort the performance of volitional acts and disturb the attitudes and postures of rest.

Dr. Charles K. Mills, Philadelphia, said that a decided advance has been made as regards the lenticular pathogenesis of cases similar to these brought brfore the society. The facts that were presented seemed to endorse more or less the belief that there is an association in the lenticular nucleus of impulses which are differentiated in the cortex. The remarks made by Dr. Sachs about the operations of Horsley and himself were possibly best interpreted along the lines of this theory. The choreic and athetoid movements, involuntary emotional expression, and various other phenomena which arise from destructive lenticular diseases, in line with the principle enunciated by Dr. Jackson, are not active symptoms of the diseased part. Disease of the lenticular nucleus, because of the associations of this ganglion with the cortex, allows a freedom of movements necessarily aberrant stimulated by these cortically represented centers.

Dr. Gustave Roussy, Paris, France, said that he had the privilege of studying the extremely interesting case reported by Dr. Spiller in which the lesions were strictly limited to the putamen. The important thing in this case was the origin of the spasticity and contracture with increased reflexes and the Babinski sign, in the presence of complete integrity of the pyramidal tract at all levels. This finding was an apparent paradox if it is remembered that Dr. Babinski insisted, from the very start, that the reflex indicated organic or functional perturbation of the pyramidal tract. Several years ago, in studying thalamic lesion with his teacher, Professor Déjerine, the Babinski reflex was never observed in cases with lesions strictly confined to the thalamus.

Dr. Israel Strauss, New York, called attention to the moving pictures illustrating the cases of dystonia musculorum, in which in the hysteria case a most remarkable wavelike motion of the thigh occurred. In the last case of the dystonia group, the boy in whom it was said there was some improvement, a somewhat similar although not as intensive wavelike motion of the thigh was present. When he was admitted to Dr. Strauss' service at the Montefiore Home the diagnosis between dystonia and hysteria was not clear. When this patient bent forward, his back would curve normally, but when erect he had a marked lordosis. His arms were likewise affected. He improved greatly by reeducation but in the beginning his walk was very much worse. The curious dystonia movements of one arm also disappeared when he was sent to the workshop. The boy, strangely enough, could run very well. Dr. Strauss was still not in a position to say whether this case was one of true dystonia or hysteria, but he thought it worth while calling attention to the curious wavelike motion that was present. It is really a very important contribution of Dr. Tilney and Dr. Goodhart if this method of differentiation could be used in these cases, and if the wavelike motion could be utilized in differential diagnosis.

Dr. J. Ramsey Hunt, New York, said that in this discussion of the striatum he would recall briefly the interpretation of its function and symptomatology which he formulated in his study of paralysis agitans (*Brain*, 1917, p. 58) and which he thought clarified many of the difficulties of the situation. The corpus striatum contains two types of nerve cells: (1) A large motor cell which is found throughout the whole of the striatum, in the globus pallidus and also in the caudate nucleus and putamen of the globus pallidus type. This is the cell type which undergoes atrophy in paralysis agitans. (2) A smaller type of cells which are quite numerous in the neostriatum. These cells undergo atrophy in Huntington's chorea, the large globus pallidus cells being well preserved. There are, therefore, two fundamental cellular systems and two essential syndromes of the corpus striatum. (1) Chorea which is related to the neostriated system, and (2) paralysis agitans, which is related to the efferent pallidal system. When the large cells of the pallidal system are destroyed, the paralysis agitans syndrome results. If the small inhibitory and coordinating cells of the neostriatum are affected, there is chorea of the Huntington type.

There are other types of pathologic lesions which are more massive in character, and which may affect the two systems in different degrees, and that is the reason we have so varied a symptomatology in affections involving the striatum. The symptomatology of Wilson's disease, dystonia musculorum, and double athetosis, simply represent degrees of involvement of these two essential systems.

Dr. William G. Spiller, Philadelphia, said he could not make any statement in regard to the Babinski reflex. The woman showed constant athetosis and the spastic condition was so intense that it was impossible to get sufficient relaxation to test this reflex.

Dr. E. W. Taylor, Boston, in response to a question as to whether the involuntary movements persisted during rest, said that it was noted in this as in other cases of similar character that these movements ceased entirely when the patient was sleeping or was not making muscular effort. The intention type of movement, if one may use that term, was very definitely defined. There were no electrical alterations and no muscular atrophy. In the moving pictures which Dr. Goodhart had shown the peculiar torsion movements which have been described were not evident. Unfortunately in the case which he himself presented, the slow moving pictures did not show the torsion movements but these were very evident in the rapid pictures. The patient in attempting to walk would often fall suddenly into a distorted position requiring him to proceed backward for a distance before he could again turn himself to face in the direction in which he desired to go. Mendel reported Thomalla's case as a doubtful one and Dr. Taylor was inclined to agree with him. The essential features were: marked involvement of face with disturbance in speech and deglutition; Babinski sign not always negative; associated athetoid movements. There were marked changes in the putamen, which was reduced in size on both sides and softened, but the nucleus caudatus and internal capsule were unaffected. The globus pallidus and the island of Reil were not involved. There was some enlargement of the thyroid, and the liver presented changes similar to those found in Wilson's disease and pseudosclerosis.

SUBTENTORIAL HERNIATION WITH HEMIANOPSIA. Dr. Adolf Meyer, Baltimore. This article appears in this issue.

A CLINICAL AND ANATOMIC STUDY OF A VASCULAR LESION LIMITED TO THE CEREBELLAR HEMISPHERES. Dr. Walter F. Schaller, San Francisco. This will appear in an early issue.

This paper discusses: marked asynergia, cerebellar catalepsy and scanning speech, absence of dysarthria, nystagmus, and of spontaneous errors of pointing after Bárány. It includes a demonstration of serial sections of the medulla, cerebellum, midbrain and basal ganglions by means of the epidiascope. It also discusses anatomic findings.

DISCUSSION

Dr. Charles K. Mills, Philadelphia, said that the case seemed very interesting in connection with the efforts recently made to determine the symptomatology and localization of destructive cerebellar lesions. Everything in the case pointed to the synergic function of the cerebellum, and there was nothing to indicate that the cerebellum is an organ directly connected with tonus. Catalepsy in cerebellar cases is rare and he had only seen one case, a man still living—in the wards of the Philadelphia General Hospital, who exhibited for a short time a sort of pseudocataleptic condition. This phenomenon of cerebellar catalepsy is not actually caused by any change in the cerebellum itself but is due to the exercise of aberrant cerebral influence. The degenerations in the crus and other degenerations here shown in connection with a study of the symptoms, enforce the idea of the synergic function of the cerebellum.

Dr. LaSalle Archambault, Albany, said that the sections were beautifully stained and very well interpreted, but it seemed to him that it was extremely difficult to interpret the symptomatology observed in this case merely from the standpoint of the cerebellar lesions. Although the main lesions were found in the cerebellar hemispheres, there were a number of small foci throughout the brain stem, one in the pontine tegmentum, another presumably in the inferior olive, and a third in the crus cerebri. All of these lesions were small, and if they had been in the cerebral hemispheres it might not have meant so very much, but when located in the brain stem where so many nuclear complexes and fiber tracts are found, they might have a great deal of bearing on the incidence of the symptoms. Dr. Archambault did not believe that there was absolute justification in attributing the various symptoms to the foci in the cerebellum. The latter lesions, while not equal on the two sides, were symmetrical, and that may account for the fact that additional functional deficits were not found.

Dr. W. W. Schaller, in closing the discussion, said in answer to Dr. Mills, that this patient had no remarkable change of muscle tonus. He would designate cerebellar catalepsy in this case as a symptom of cerebellar disease without attempting to explain its exact mechanism. The objections of Dr. Archambault were explained on the basis of secondary degeneration occurring in the olive and the median fillet; the lesion in the median fillet and lateral fillet would hardly have any important bearing on the motor symptomatology of the case.

ELEMENTS OF PREDISPOSITION AND DETERMINING CAUSES OF SECONDARY DISORDERS IN PSYCHONEUROTIC ACROPARALYSIS RESULTING FROM THE WAR: THE DYSKINETIC SYNDROME. Dr. Gustave Roussy, Paris. This article appeared in full in the September issue, page 247.

An unusual syndrome with peculiar clinical manifestations was brought to light by the war. It is characterized by contractures resulting in fixation of the large articulations in defective postures, and by special motor difficulties (contraction, paralysis, or both) of the distal ends of the limbs. Some time following nervous shock or slight traumatism, impotency develops on a pithiatic basis and the immobility or defective function leads to organic accidents. This is a hystero-organic condition and must be considered in its elements: (1) the chief motor disorder, and the secondary (vasomotor, trophic, etc.) disorders. The motor disorder, the primary element, is of pithiatic character, capable of reproduction at the will of the patient and liable to disappear suddenly under favorable suggestion. The associated disorders, of variable character and very persistent, are comparable to admittedly hysteric disorders and are functional accidents based on predisposition to pithiatic motor disturbances. Investigation has shown that in predisposed patients there is marked bilateral vasoconstriction and microsphygmia; if hyperhidrosis or musculo-tendinous laxity is present this will also be apparent on the unaffected side, though less marked. Special predisposing associations lead to the production of articular disorders and of strong reflex reactions. Through psychic inertia, passive or forced immobility may aggravate preexisting tendencies, and under the influence of a special neuropathic turn of mind and perverted will the persistent suppression of function causes the development of the secondary disorders. Restoration of activity leads to improvement.

DISCUSSION

Dr. Smith Ely Jelliffe, New York, said that the matter of extreme interest to him was that Dr. Roussy had emphasized clearly and had well illustrated that the emotional background, or the emotional dynamic factor, was the paramount situation in practically all of these cases. There have been many discussions on this subject. Dr. Jelliffe was a believer in the intrinsic effects of emotional situations, and had emphasized frequently that many disease pictures which have heretofore been called organic start at purely emotional levels, and ultimately, through long fixation and by long unconscious repression of affective material, arrive at organic goals. It was gratifying to hear that after a long period of time, eighteen months in one case and seven months in another, the hypertrophic changes in the skin, nails, sweat glands, and hair cleared up through rational psychotherapy. Dr. Jelliffe thought it remarkable that in so short a time such splendid results were obtained with this type of clientele. It is a matter of extreme regret, on the other hand, to recall how refractory so many similar conditions had proved in some of his own patients. And this stimulated him to attempt to arrive at some deductions concerning the mensuration of these emotional forces, about which Dr. Roussy mentioned certain experimental measurements of the amount of perspiration, and of oscillations of blood pressure conditions.

Dr. Francis X. Dercum, Philadelphia, said that it was noted by the English physicians that soldiers presenting functional nervous symptoms usually improved when sent to England, but relapsed if ordered back to the front, and at times, even before they got to the channel. In this connection the report of Surgeon-General Ireland, that out of 2,300 cases of functional neuroses—shell shock—2,000 recovered on the day of the armistice, is very significant. It would appear that when all prospect of reentering active service disappeared, the cases recovered. These cases had a parallel in civil life in the cases of hysteria following accidents, in which there were claims for damages. No improvement results until the question of damages has been disposed of, in one way or another. He understood from Dr. Roussy that the soldiers who were treated successfully were men who had not been in active service for some two years and who had practically no prospects of returning to the trenches.

Dr. William G. Spiller, Philadelphia, said that the pictures which Dr. Roussy had shown were exceedingly instructive. Especially interesting was the method of making the paralyzed hand move synchronously in the same kind of movement as that performed by the unaffected hand. A similar result might be obtained in organic conditions. This method of treatment may be employed after paralysis from a lesion of the central nervous system, or where there has been recovery from paralysis without return of function despite removal of the cause of the paralysis. Oppenheim called this condition akinesia amnestica. The nervous system seems to have forgotten how to make use of its function, if it may be so expressed. Dr. Spiller had at the time a case of peripheral facial palsy in which the benefit of this method of treatment was shown. The man had a severe organic facial palsy. He was made to draw up the nonparalyzed corner of his mouth, and each time he did so the galvanic current was passed on the paralyzed side of the face, and considerable power had been regained.

Dr. Daniel J. McCarthy, Philadelphia, believed a slightly different interpretation could be placed on these cases than that which Dr. Jelliffe gave. In a study of a number of cases of this type in 1914 and 1915 at the American Ambulance, and in 1918 and 1919 at the Neurological Institute at Vichy, a definite number of cases fell into a group which explained to a certain extent the bilateral symptoms. He assumed that in cases in which there was very intense injury, for example, there were physiologic reactions not only on the same side but on the opposite side as well, and in studying a series of intense injuries of the bones of the shoulder girdle it was found that particularly after these accidents there were trophic disturbances on both sides. In rare instances there was clear cut delimitation of function and sensation confined to the injured side. It is a vast problem, and it should be remembered that the question of a separation of what might be called functional overflow and actual organic disease must be dealt with. Dr. McCarthy believed that in this type of case there is a definite reaction with certain vasomotor and trophic disturbances, which, if the individual had sufficient purpose for remaining out of the service and if he was not properly examined in the beginning, would have become apparently organic. At least this condition obtained in Dr. McCarthy's service. There were approximately 1,000 cases at the Institute of Vichy, but very few of the intensity of Dr. Roussy's cases, because the patients realized that a clear cut elimination of the functional group was made, and therefore the symptoms cleared up. Those cases which one might interpret as the segmental group, with intensive injury of nerves, muscles, or bones and clear cut symptoms on the other side, did not prove persistent, except in a few instances. Dr. Roussy's presentation was extremely interesting but the interpretation is open to question. The primary condition is first organic, and secondly reflex on the vasomotors, affecting rarely the opposite side. What might be called hysteria, or defense reaction, kept the individual out of the service with persistent symptoms. Hysteria must here be used in the broadest sense of the term. Any man who has been through front line service would not be glad to return, and if he knew that an injury of the hand would be a decisive factor in postponing his return, he would try to prolong his symptoms until the end of the war. On the other hand, in contradistinction to such cases, there were those soldiers who, as soon as they found that they were dealing with diagnostic tests, realized that they would have a difficult time of it, and got well.

Dr. Gustave Roussy, in closing the discussion, said that in regard to the question brought up by Dr. Dercum, it was a very difficult matter to give absolute statistics or percentages, but approximately in from one-tenth to one fourth of the cases, there was actual failure, either partial or complete, in obtaining therapeutic results. The remaining three fourths could again be subdivided; some of them were completely cured and returned to active service, but the greater number had already had disturbances for several months or years. These soldiers were not discharged entirely, but were placed in what was known as the auxiliary service and kept under military discipline. The next point emphasized was that the various neuroses which developed during the war were psychologically analogous to those observed in civil life, but in interpreting the ease with which the therapeutic result was attained, one had to remember the extraordinary prestige and influence of the military physician, who was not only a physician, but likewise a military officer. Furthermore, everything was so arranged that all cases could be grouped under one roof, so to speak, or in one fairly well compassed area subject to the general supervision of one medical and military head, so that all privileges and disciplinary measures were absolutely regulated. The men were practically obliged to submit integrally to the various measures adopted for the relief of their symptoms. Unquestionably some of these cases were cured much more promptly after the cessation of hostilities, but an important fact was that even at that time a large number of patients failed to recover spontaneously. They had to be helped by some therapeutic measure. In regard to Dr. McCarthy's question as to whether the various motor disturbances were not primarily organic and secondarily functional, the opposite view had been assumed by him, by reason of the experiences which he had had, namely, that the motor disturbances were functional from the outset, inasmuch as if treated at once they were invariably cured completely and no secondary trophic or vasomotor disturbances appeared.

REPORT ON THE STUDIES OF DR. R. S. ELLIS TOUCHING THE STRUCTURAL CHANGES IN THE HUMAN CEREBELLUM—FROM BIRTH TO OLD AGE—TOGETHER WITH A QUANTITATIVE DETERMINATION OF THE PURKINJE CELLS IN NORMAL AND SUBNORMAL CEREBELLA. Dr. Henry H. Donaldson.

Between birth and 15 months the external layer of granule cells disappears and this event coincides with the appearance of the ability to walk. The relative weight of the human cerebellum characteristic for the adult is attained at about 15 months, and this relative weight is not significantly modified by stature, sex, age, race or grade of normal intelligence. After the thirtieth year there begins to appear a systematic loss in the number of Purkinje cells and this becomes more marked in old age. There is some evidence to show that this loss is usually greater in the right cerebellar hemisphere. In idiots and imbeciles this loss is very marked and in them some correlations have been made between the localities of the greater deficiencies and the type of motor disturbance.

DISCUSSION

Dr. Harold N. Moyer, Chicago, asked if there was any relation between right handed and left handed individuals in the results obtained in these studies.

Dr. Edward D. Fisher, New York, asked what the relative difference was between the cerebral cortex and the cerebellum in regard to the disappearance of cells as individuals grow older.

Dr. Donaldson, in closing the discussion, answered that he had no data to present on the cerebral cortex. As to Dr. Moyer's question, unfortunately he had not the records as to whether the individuals were right handed or left handed, but he assumed that right handed individuals predominated. He added that the volume of the full Purkinje cell was attained in about two years.

RARER CLINICAL TYPES OF EPIDEMIC ENCEPHALITIS. Dr. Bernard Sachs. This paper will appear in an early issue.

This paper is a presentation of some of the more unusual forms of epidemic encephalitis, with special reference to those presenting meningeal and psychic symptoms. A few other cases are also given to prove the justice of speaking of these epidemic cases as "central or basilar encephalitis."

FURTHER STUDIES ON THE ETIOLOGY OF ACUTE ENCEPHALITIS. Drs. Israel Strauss and Leo Loewe.

Cultural characteristics of the organism. Animal inoculations. Study of the nasal washings and spinal fluid and the use of these as a means of diagnosis.

THE TENDENCY OF EPIDEMIC ENCEPHALITIS TO CHRONICITY. Dr. Isadore Abrahamson. This paper appears in this issue.

CHOREO-ATHETOID AND CHOREOPSYCHOTIC SYNDROMES AS CLINICAL TYPES OR SEQUELAE OF EPIDEMIC ENCEPHALITIS. Dr. LaSalle Archambault. This paper will appear in an early issue.

Epidemic encephalitis is characterized by a protean symptomatology, the variations of which are dependent on the location of the predominant histologic lesion. In addition to the usual and familiar clinical picture of midbrain involvement, the disease may be manifested in a number of clinical types closely resembling such diseases as paralysis agitans, poliomyelitis, polio-encephalitis, polyneuritis, cerebellar sclerosis, hemiplegia and others. Aside from the disparity in clinical manifestations, there are variations in the course and evolution of the disease. The disease may be of acute or subacute onset, and its duration may be very variable, even to extending over several months. After the complete disappearance of a few abortive manifestations of typical encephalitis, certain choreiform twitchings and acute psychotic disturbances are not uncommonly seen as the salient manifestations, and choreo-athetoid or frankly athetoid syndromes may be observed as sequelae of the disease. The occurrence of a chronic or relapsing form of epidemic encephalitis may have an important bearing on the origin of some of the affections simulated by the clinical types of the disease and of such obscure conditions as multiple sclerosis, hypothalamic and dysglandular syndromes, and certain psychoses, including perhaps dementia praecox. The variations in the symptomatology are possibly due to individual variations in the relative susceptibility of different levels of the cerebrospinal axis; regional exhaustion from prolonged hyperactivity or previous disease or trauma is an important factor in the localization of the lesions produced by the infection of the central nervous system. To this same factor may be ascribed the frequent involvement of the mesencephalon in epidemic encephalitis with its resultant lethargy and oculomotor paralysis; probably never before have people slept so little and used their eyes so much as within the last five years.

DISCUSSION ON PAPERS OF DRS. SACHS, STRAUSS AND LOEWE, ABRAHAMSON AND ARCHAMBAULT

Dr. Daniel J. McCarthy, Philadelphia, in reference to the incidence of encephalitis in this epidemic as compared with the epidemic of 1889-1890, in contradistinction to Dr. Sachs' experience, recalled that in those days there was a very definite group of cases not described as sleeping sickness, or as encephalitis, which conformed very closely to the type seen at the present time. In those days, the cases came to attention two, three or four years after the original epidemic. There were milder epidemics during the following two or three years. As he remembered them, they were very often of a severe type with spinal symptoms.

With reference to diagnosis, in the Army in France there was an epidemic of influenza, but it was not as intense as the epidemic in this country. He remembered Dr. Cushing calling his attention to the ephemeral nature of the early ocular cerebral symptoms. The ocular palsies were very transient and lasted only one, two or three days, and then sometimes returned accompanied by definite somnolence and more fixed symptoms. In those cases the principal feature was the extremely slow development of the symptoms; a patient might go along for two, three or four weeks before the diagnosis was finally established and he reported sick for examination. He had a very slight attack of this condition himself and went through a period of somnolence with only one symptom of cortical irritation affecting the muscles of one hand. As one follows the pathology of this disease there is no necessity, from his standpoint, to make any remarkable divisions of the distribution of the lesions in epidemic encephalitis, since these distributions may be extensive and widespread, and in that way leave a general picture of the disease as one sees it. The diagnosis can be very simple without an arbitrary classification.

Dr. Adolf Meyer, Baltimore, asked for the opportunity of demonstrating a few lantern slides which would direct attention to one variety of the disturbance that has not specifically been named—pseudotumor. They dealt with a patient who showed symptoms suggesting encephalitis in the early process of the disease, and who, about three months afterward, began to show some head symptoms—choked disk, complete blindness, complete deafness, with a certain amount of staggering. The patient died during an operation for decompression, and showed, in reality, a diffuse meningitic process, with a peculiar type of cell infiltration. With all these apparent pressure symptoms and the tremendously diffuse meningitic and encephalitic process, the patient showed a perfectly astounding mental clearness and ease.

The brain showed practically no evidence of macroscopic disorder, except a little asymmetry and hemorrhagic discoloration of the uvula of the cerebellum and the choroid plexus and slight milkiness of the pia of the convexity of the cerebrum. Under microscopic examination there were found a peculiar infiltration of the meninges of the base, the cerebellum and the cerebrum, and a few encephalitic foci in the hind-brain and cerebrum, with a large cell type, reminding one of the cells Essick found under mild irritation of the pia arachnoid, only much larger, often pear-shaped and epithelioid, with total absence of the characteristic small lymphoid type of infiltration. The chief points in the case were the symptomatology of pseudotumor with choked disk, deafness, blindness, remarkable mental clearness of the patient and the diffuse meningitic infiltration with this peculiar cell type; hence the propriety of speaking of a meningo-encephalitis magnocellularis.

Dr. Harold N. Moyer, Chicago, said that the remark of Dr. Sachs regarding the ease with which a correct diagnosis may be made during an epidemic suggested that a wrong diagnosis may be made with equal facility. In a well marked case reported by Dr. Bassoe presenting a typical picture of this disorder, including the temperature changes, was found to be a case of tumor of the midbrain. A patient in Dr. Moyer's service at the Psychopathic Hospital presenting a typical clinical picture of the lethargic form was found after death to be suffering from a basal skull fracture.

Dr. Colin K. Russel, Montreal, said that one of the very interesting points in this epidemic has been the symptom of somnolence, chiefly because, in his experience at least, it was practically seen only in cases where the clinical evidence pointed to a lesion in the posterior part of the pons, giving rise to ocular symptoms, particularly nystagmus. He had seen this symptom also in a young patient in whom the clinical evidence of laryngeal paralysis and paralysis of the muscles of the neck pointed to a lesion in the lower part of the medulla and upper cervical cord. Somnolence in this case was marked and lasted over a week. On the other hand, in a patient with an apparently uncomplicated bilateral facial paralysis, there was no history of somnolence. The interesting suggestion had been offered that this symptom might be due to involvement of the pituitary gland, but in one of his necropsies, in a case with marked somnolence no pathologic lesions of this organ were found. Dr. Russel's experience rather inclined him to agree with Farquhar Buzzard's suggestion that the somnolence might be due to a closing off of the aqueduct of Sylvius giving rise to disturbances in the circulation of the cerebrospinal fluid and an acute hydrocephalus.

Dr. William G. Spiller, Philadelphia, said that the short duration of the ocular palsies was characteristic. They come rapidly; in one patient the first symptom noticed was double vision in looking out of a window. For this reason the ocular palsies might be overlooked. This is also true of other cranial palsies. He had seen facial palsy lasting only a few days. Regarding Dr. Archambault's observations about the choreic movements, Dr. Spiller said there was a patient in the University Hospital, sent by Dr. George Wilson, who had had these choreic movements since the epidemic of 1918. Dr. Spiller had come to believe, although possibly he had not sufficient basis for this conclusion, that the choreic or myoclonic form of lethargic encephalitis was caused by a lesion in the region of the basal ganglions rather than by a lesion near the aqueduct of Sylvius. He referred to that in his paper before the Association the previous day. Dr. Spiller states he did not know of any necropsies that had been reported in the myoclonic type. He had a case in March of this year of a man who had had this myoclonic type for several weeks. He admitted him to the hospital, and not supposing there was any immediate danger, expected to see him the next morning, but before he arrived the patient had died. In the necropsy he found that the perivascular infiltration was slight in the region of the aqueduct of Sylvius, but that there was considerable infiltration throughout the basal part of the temporal lobe.

In regard to the somnolence, this symptom seems to be more striking in cases in which the region around the aqueduct of Sylvius is involved. It has been the view of some that the optic thalamus is the recipient of all afferent impulses, and when it is diseased the impulses are unable to reach the cortex and lethargy results.

About October of 1918 Dr. Spiller had a case in his service which was reported by his intern, Dr. Smith, and published in the Archives of Neurology and Psychiatry, and which seemed at first to be one of tumor. It was the case of a young boy who had choked disk, ocular palsies and a paralysis of the soft palate. It was very difficult to explain this combination by a diagnosis of tumor. It was not tumor and complete recovery occurred. The case was regarded as one of encephalitis simulating tumor, and Dr. Spiller referred to it later in the same journal under this diagnosis.

Dr. Israel Strauss, New York (in closing discussion), said for both Dr. Sachs and himself, that their experience at the hospital covered a total of something over a hundred cases, and he had seen in private practice at the lowest estimate about fifty additional cases. They have observed practically every form of this disease. The French have begun to describe a large number of syndromes, but all of these have been observed in this country.

They have also observed the transient palsies that Dr. McCarthy mentioned, and which they regard as abortive types. Regarding the cases following the influenza epidemic of the nineties, Dr. Strauss said he could not speak from personal experience, but he would like to call Dr. McCarthy's attention to the fact that at that time a great number of cases of acute encephalitis were described and were supposed to be influenzal encephalitis. In their work they found one strain, which in the beginning produced the picture of hemorrhagic encephalitis, and as they transmitted that virus through animals its character changed so that it produced the histologic picture now regarded as typical of lethargic encephalitis.

Another interesting fact noted was that in one of the cases of an acute maniacal state, with myoclonus, the organism was obtained. Strangely enough, those patients sometimes, without any symptoms of palsy, were the ones that died very soon.

Dr. Strauss stated that he thought it hazardous to draw conclusions regarding the site of the region involved in symptomatology, from the extent of the lesion found in any part of the brain. They had performed a large number of necropsy examinations and had been struck with the fact that in some cases the extent of the lesion was remarkable, the entire basal ganglions being affected in toto, while in other cases, showing precisely the same symptomatology, just as severe in type, they were amazed to find such a small lesion. Dr. Sachs had referred to the fact that one can account for the severity of the disease by the severity of the lesion. For this reason he believed it would be hazardous for anyone to postulate on what is found in the brain any theory regarding the localization of any given symptoms.

Dr. LaSalle Archambault, Albany (in closing), said that from the beginning the papers published on this disease had given a rather confusing impression of the condition of the spinal fluid. Many series of cases have been published in which the fluid had been almost universally negative. Dr. Archambault said that had not been his experience at the Albany Hospital. Definite changes were found in the majority of cases. He was unable to state the exact percentage, but it was possibly 60 to 70 per cent. The findings were not pronounced, but definite. The cell count varied from 8 to 200 cells per cubic millimeter, and the globulin was positive, the reaction being reported as two or three plus. In some cases there was a peculiar reaction with the colloidal gold test; the discoloration occurring in the first tubes somewhat suggested the findings in syphilis or general paresis. The Wassermann reaction was negative in both the spinal fluid and the blood.

As Dr. Strauss had brought out, there were many cases diagnosed as encephalitis, and other cases which remained undiagnosed. Eventually we shall have more accurate means of diagnosis, but in the meantime we are not all fortunate enough to have Dr. Strauss with us, and in the absence of such positive scientific data as he would be able to furnish, other available means must answer. Dr. Archambault believed and would like to go on record as stating that, while not absolutely essential or necessary, changes such as he had described in the spinal fluid were of extreme value in helping to make a proper diagnosis in epidemic encephalitis.

CEREBRAL SURGERY. Dr. Harvey Cushing.

In two interesting and suggestive papers Weed and McKibben[1] demonstrated the significant physiologic fact that it is possible to reduce the cerebrospinal fluid pressure and diminish the bulk of the brain by injecting a hypertonic solution into the blood stream. Conversely they found that hypotonic solutions had the opposite effect: a rise of cerebrospinal fluid pressure and an increase of brain bulk. In the course of our studies their work has been repeated and their general conclusions have been confirmed. The clinical bearing of these facts is obvious. They concern particularly the states commonly referred to as "pressure symptoms." By similar methods it has been found possible to secure the same results in patients with increased degrees of intracranial tension. It was felt that the undesirable effects on pulse, respiration and blood pressure of such intravenous injections might contraindicate their use. For this reason the effects of gastro-intestinal doses of hypertonic solutions were studied.

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[1] Experimental Alterations of Brain Bulk, Am. J. Physiol. **48**:531, 1919; Pressure Changes in the Cerebrospinal Fluid Following Intravenous Injection of Solutions of Various Concentrations, ibid., p. 512.

In a large series of animal experiments it has been found that practically the same effects may be obtained by gastro-intestinal administration. By this method the intracranial changes are not attended by disturbances of pulse, respiration or blood pressure, and the possible alterations of the cellular elements of the blood are avoided. From 20 to 30 c.c. of a saturated sodium chlorid solution introduced into the duodenum or rectum of an average-sized cat produced a maximal fall of cerebrospinal fluid pressure precisely comparable to that which occurred when the solution was given intravenously. Following such doses the average fall of pressure in a large series of experiments was 258 mm. normal saline. The changes were roughly proportionate to the concentration of the salt and the size of the dose. Two per cent. sodium chlorid solution in large doses or a saturated solution in doses as small as 5 c.c. gave corresponding though less marked effects. Nonabsorbable salts gave similar reductions though slower in occurrence and less in extent. Dextrose solution caused results still less striking though identical in nature. The converse results seen after the ingestion of hypotonic solutions (water) were not great in extent nor well sustained. A previous dose of a hypertonic solution rendered more extensive the rise of pressure in these cases. The changes observed were independent of blood pressure and were not attended by significant alterations of pulse or respiration.

These effects on cerebrospinal fluid pressure and brain volume were investigated in human subjects, an excellent opportunity for this being afforded by patients with brain tumor and cerebral hernias subsequent to decompression operations. In these patients there was a lowering of tension when hypertonic saline solutions were given by mouth. Occasionally very striking results could be obtained, in which cases the tense convex protrusion became a soft concave area over the decompression site.

PUNCTURE OF THE CISTERNA MAGNA. Dr. James B. Ayer. This paper will appear in an early issue.

DISCUSSION OF PAPERS BY DRS. CUSHING AND AYER

Dr. Ernest Sachs, St. Louis, said that he had come to realize in the last few years that the handling of the cerebrospinal fluid was one of the most important factors in dealing with intracranial conditions, particularly tumors. Consequently, he had been greatly interested in the work of Weed and McKibben. Dr. Cushing pointed out in his paper that he had shown experimentally that saturated salt solution injected intravenously would reduce the volume of the brain. Dr. Sachs had tried this on several cases, just as Dr. Cushing had done. The result in the reduction of the hernia was so striking that one of his assistants, Dr. Belcher, urged him to try it in the course of an operation. Since he had only Weed's experimental evidence to go on, he hesitated to use it except in a case in which it was impossible to get cerebrospinal fluid by ventricle puncture, as happens not infrequently in supratentorial tumors, because these tumors often compress the ventricle. In these cases opening the dura without reducing intracranial pressure is likely to meet with disaster. He recently tried this, therefore, in the course of an operation and it gave satisfactory results, and although it was only a single observation, it seemed worth recording as the method promises to be of great value.

This particular case was one of temporal lobe tumor in which both ventricles were aspirated during operation to reduce pressure, prior to opening the dura, and in which both the right and left ventricle were completely compressed and only a drop or two of fluid was obtained. There was then introduced, intravenously, 30 to 35 c.c. of a 35 per cent. saturated solution of salt and within five to six minutes the brain had contracted to such an extent that it was possible to open the dura with the brain practically under normal tension. Studies were made in this case for a number of days afterward to determine whether there was any harmful effect from the salt solution and also whether the fragility of the red blood cells might have become affected, a point that Dr. Marshall, our pharmacologist, was much interested in. The studies made in this one case showed that the fragility of the red blood corpuscles was not impaired in any way, and as far as they were able to observe, there was no deleterious effect on the patient.

It seemed to Dr, Sachs that this procedure during the course of intracranial operations was one that must be undertaken. He undertook it with much hesitation, but felt that the experimental work of Weed and McKibben justified it in an extreme case.

Regarding the work of Dr. Ayer, he felt as most surgeons did who did not like to undertake procedures in the dark. He would fear greatly to undertake such a puncture of the brain cisterna. He noticed in his drawing that Dr. Ayer did not take into consideration two points which, it seemed to him, must be remembered: First, the medulla is suspended in fluid and, therefore, during life it may be much nearer the posterior portion of the canal than it is in the cadaver. The cord and medulla move markedly during respiration. During inspiration the cord is forced back, as can be seen in the course of any spinal operation. And, second, not infrequently there is a vein of considerable size that runs from the torcular down the median line in the dura, and Dr. Sachs thought there would be great danger in some cases of puncturing that vein since, according to Dr. Ayer's drawing, the needle is passed so close to the posterior edge of the foramen magnum that where this vein ends in the circular vein around the foramen it may readily be injured. No one cares to have blood in the region of the medulla; surgeons try to avoid having even a drop there.

Dr. Harvey Cushing, Boston, thought there could be no doubt but that Dr. Ayer had realized the risks of this procedure as much as any one.

In regard to what Dr. Sachs had mentioned, Dr. Cushing said that this particular median vein stopped at the foramen, so that there was no risk on that score. Nevertheless, the procedure is a dangerous one. However, he cautioned that Dr. Ayer was merely reporting some preliminary studies, and even lumbar puncture under certain circumstances is dangerous. He was quite sure that the procedure which Dr. Ayer had in mind may, with due precautions, become useful. He thought the same thing was true of the saline procedure, of which he had spoken.

Dr. Cushing said it must be realized that we are in the early stages of these investigations, and it was long after Quincke's time that lumbar puncture became routine. He was sure there is a great deal in all of these new procedures that may become valuable, but he believed that such a measure as Dr. Ayer advocated should not be promptly adopted by all physicians, as accidents would follow. The measure should be left in the hands of those who were particularly interested in it, to develop the technic until we could take it over from them and rely on it. He thought the same thing was true of injecting air into the ventricle for diagnostic purposes. The time had not yet come when any one of these three novel procedures could be advocated for general use.

Dr. Foster Kennedy, New York City, said that Dr. Ayer's description of the first step in his technic and his directions for finding the first cervical vertebra rather reminded one of the opening sentence of Mrs. Beaton's recipe for making Welsh rarebit—"First catch your hare."

The procedure that Dr. Ayer had touched on was one that ought to be viewed somewhat askance, and as Dr. Sachs wisely pointed out, its technic ought to deter us from undertaking it lightly. Surgery in the dark, as Dr. Sachs remarked, is, on the whole, rather a precarious procedure, and when in the neighborhood of the medulla, since the medulla is not a fixed point, it seemed to Dr. Kennedy to be doubtful. If it is a matter of removing cerebrospinal fluid from the great cistern of the brain, and one has the choice of tapping the cistern, or giving saline by mouth, it seemed better for the patient to take the salt!

As for the suggestion that this method is a proper one with which to treat cerebrospinal syphilis, he said that this has not yet been demonstrated.

Dr. Adolf Meyer, Baltimore, called attention to a way of getting a good view of this region with a procedure that suggested itself to him because he desired to obtain more perfect specimens at necropsy. The attempt to obtain a well preserved position and normal topographical relations of the brain led Dr. C. B. Dunlap to work out a technic of formaldehyd injection before removal of the brain at necropsy. This made of the brain a relatively inelastic mass; hence the necessity of adding to the usual circular section of the skull the removal of a wedge of the occipital bone, exposing the foramen magnum and under the dura the pia arachnoid of that region.

Having thus had the frequent opportunity of seeing that region in formaldehyd injected material under the best possible exposure, so that we were not in the dark, Dr. Meyer was decidedly impressed with the fact that we were dealing with a space much wider and more easily reached than he would have dreamed. The tapping of the posterior tent of the pia arachnoid was, really, as Dr. Ayer described, much safer and easier than any one who had not performed that necropsy technic would consider likely. He thought it wise, however, to consider the possibility of cerebellar herniation.

Dr. Bronislaw Onuf, Rutherford, N. J., wished to state that he had applied Dr. Ayer's method in about fifty cases with entire safety, meaning that as it was used postmortem, the danger of causing death was excluded.

He had previously referred to a method which he wished to speak of again because the subject had some bearing on Dr. Cushing's paper. Some fifteen years ago he described a method of preserving the brain and spinal cord after death without performing necropsy. He was, at the time, pathologist to Craig Colony, and in many cases he had to wait twenty-four to fifty hours after death for permission to perform the necropsy examination. In most of these cases the brain and cord would have been absolutely annihilated for microscopic study if something had not been done to preserve it before the necropsy was made. A method was devised which was described in the *Medical Record* (July 9, 1904) of injecting a solution of formaldehyd, 12 per cent., through two routes—one by lumbar puncture and the other through the cisterna magna. The injection was made under pressure, and he was surprised at the large amount of fluid that could be injected in this manner. Usuaully, only about 400 c.c. could be so injected, but he had had cases in which as much as 1,500 c.c. went in. The fluid oozed out along the nerve roots, hardening the parts adjacent to the spine, lungs, liver, kidneys, intestines, etc. That the fixation was a success was shown by applying to the basal ganglions such delicate tests as Weigert's and Mallory's neuroglia stains which succeed only in specimens hardened almost immediately after death, and which in the specimens made from the basal ganglions of brains hardened by the prenecropsy method mentioned, gave perfect results.

The point of interest with regard to Dr. Cushing's investigations was the oozing out of the fluid along the nerve roots, showing some of the pathways along which the cerebrospinal fluid may wander.

Dr. Israel Strauss, New York, thought we ought not to allow this piece of work of Dr. Ayer to pass without some commendation instead of criticism. Dr. Ayer had not only experimented on animals—eleven hundred—but on the cadaver, and his results were based on experimental knowledge.

When it came to "working in the dark" he believed that some of the procedures, like that advocated by Dr. Ayer, were safer than some others in which surgery worked in the light. He believed he would take chances with this procedure rather than allow general surgeons to attempt to aspirate the ventricles, because he had seen experts fail even in this.

As Dr. Cushing had stated, there were dangers in lumbar puncture and there were dangers in every other procedure. He had seen a patient die from an ordinary injection of one-hundredth of a grain of hyoscin, and yet this drug was in general use.

There was only one warning that ought to be given regarding Dr. Ayer's work and that was that before any of us, or the surgeons who "worked in the light," attempted this procedure, they should first try it on the cadaver. This is the particular point to which Dr. Ayer should call attention when publishing this paper, namely, asking physicians to first try it on the cadaver, and not to discredit a procedure as harmful which was of great value when properly used.

Dr. James B. Ayer (in closing) said he did not expect the acceptance of this procedure without remonstrance, and he was more than pleased that so many thought there was anything of value in it. He, too, realized the potential dangers of cisterna puncture, but believed that if used carefully there would be no harmful results.

There was another group of cases in which cistern puncture should be of use. In septic meningitis, for which there was at present no adequate treatment, irrigation from cistern to lumbar sac, or ventricle to cistern or cerebral subarachnoid space to cistern, offered a method which may well be tried. As to the serum treatment of cerebral syphilis by this route, he was not sanguine, but if there was value in serum treatment it should unquestionably be of greater value if given by this rather than by the lumbar route.

In finding the point for insertion of the needle, the landmark was not the spine of the first, but of the second cervical vertebra, which is readily palpated on most subjects. Points in technic were necessarily made brief because of the time, and because this had already been published.

The conception of the medulla being freely movable did not seem at all likely. In a cadaver in which a needle was left in situ, the brain was removed until the medulla and needle were visible; the head was then thrown into different positions; in no attitude could the needle be made to impinge on the medulla.

CHICAGO NEUROLOGICAL SOCIETY

Regular Monthly Meeting, May 29, 1920

Lewis J. Pollock, M.D., *President, in the Chair*

DEMONSTRATION OF A BRAIN WITH DEEP BRUISE AND A BRAIN WITH PORENCEPHALUS. Presented by Dr. E. R. LeCount (by invitation).

Dr. LeCount presented a brain with a deep bruise and hemorrhage which closely simulated the lesions of apoplexy, and exhibited charts showing a fracture of the right side of the skull. With these injuries there had been marked compression from an extradural hemorrhage. He stated that he had seen few deep bruises of the brain and that much bruising of the brain of any sort with extradural hemorrhage was uncommon.

The patient was a man of 25 years, and death had occurred eleven days after the injury. Dr. LeCount referred to the possibility of the deep hemorrhage being secondary, a so-called late traumatic apoplexy.

The brain with porencephalus was from a large negro, 6 feet tall and weighing 160 pounds, removed from a car on the Illinois Central during the winter with frostbitten feet and legs and taken to the Hospital of the House of Correction, where he died within a few hours, without any symptoms referable to the brain. Dr. LeCount thought that the malformation of the brain caused death because of the marked edema of its substance; moreover, postmortem examination failed to disclose disease elsewhere adequate to explain death. The brain was flattened and the frontal lobe, on which the defect was located, was adherent to the dura on the right side. When this adherent dura was lifted up a little a clear fluid, now opaque from liquor formaldehydi, could be seen which extended into the right ventricle. It is not an uncommon maldevelopment, 200 or more being reported, but so far as he knew the brains had been studied only as to the maldevelopment. In his opinion, the brain should be sectioned and studied, not for an explanation of the defect, but for its usefulness in connection with special problems of nerve pathways through the brain, still unsolved.

PARTIAL OCCLUSION OF THE RIGHT POSTERIOR INFERIOR CEREBELLAR ARTERY. Dr. George W. Hall.

A man, aged 34, married, father of one healthy child, gave no history of illness of importance; he had never had syphilis; his use of liquor had always been moderate; he smoked about six cigars a day. No history of miscarriages.

On April 19, 1920, the patient noticed a "cool" taste in the right side of the mouth. On April 20, while attempting to get on a street car he fell to the right and lost out of his right hand the money which he had ready to pay his fare. On April 23, a similar cool feeling gradually spread over the right side of his face, more noticeable on the lower lip. On April 24 he had a slight attack of vertigo when changing his posture from horizontal to vertical On April 26 he noticed some difficulty in controlling movements of the right arm. The right leg was normal. On the same day he had some difficulty in articulating and also had slight blurring of vision which cleared up within a day or two.

Examination on April 27 showed blood pressure to be systolic 125; the urine, heart and lungs were normal and there was no evidence of gastro-intestinal disturbance.

There was distinct sensory disturbance over the right half of the tongue and right side of the face and slight narrowing of the right pupil and palpebral fissure. Both pupils responded to light and accommodation. There was no diplopia, but when the patient looked straight ahead horizontal nystagmus was present and became exaggerated on looking to the right and left. There was no paresis of the facial muscles, no deviation of the jaw on opening the mouth and no disturbance of hearing; the pulse was normal. The tongue showed no deviation. Except as noted above, sensation was normal. The abdominal reflexes were normal; the knee jerks were lively, there was no Babinski reflex and no ankle clonus. When the patient attempted to rise from a sitting position there was a tendency to fall to the right; he stated that the room seemed to go to the left, but his body did not feel as though it were moving. The tendency to go to the right was exaggerated when walking with eyes closed. There was no ataxia of the hands.

On April 28, while attempting to lift a glass of water to his mouth with the right hand, the glass slipped from his grasp and fell to the floor.

The blood Wassermann was negative.

The symptoms above described remained about the same until May 3, when they gradually disappeared. On May 12 there were no numbness and dizziness. Examination on May 20 revealed nothing abnormal.

Report of Otological Findings (by Dr. J. Gordon Wilson).—Lying in bed. No vertigo. Nystagmus: horizontal, slow to right; range small; diminished if he looks to right; increased if he looks to left. Pointing: right hand, touches up down, and down up; left hand: touches up down, and down up. Rising in bed: Vertigo—feeling of instability, not rotation. Sitting up: Nystagmus and pointing as before. Hearing: Normal; no tinnitus.

Conclusions: (1) Labyrinthine nystagmus, (2) vertigo on displacement of head, and (3) no acoustic involvement.

Caloric.—April 30, 1920. Right Ear (7 ounces of water at 58 F.). Head forward 30 degrees, vertical canal: Nystagmus marked, slow to right, rotatory. Pointing: right touches; left touches. Vertigo, slight. Head back 60 degrees, horizontal canal: Nystagmus marked, slow to right, horizontal. Pointing: right touches; left touches. Vertigo slight.

May 3: Right ear (water, 20 ounces at 57 F.). Superior canal, nystagmus, slow to right; rotatory 60 seconds. Horizontal canal, nystagmus, slow to right, horizontal 90 seconds. Pointing: touches with right and left after both superior and horizontal stimulation.

April 30. Left Ear (5 ounces water at 58 F.). Head forward 30 degrees: Nystagmus, slow to left, rotatory. Pointing: right to left, 3 inches; left to left, 3 inches. Vertigo slight. Head back 60 degrees: Nystagmus, slow to left, horizontal. Pointing: left to left, 4 inches; right to left, 3 inches. Nystagmus persists longer in horizontal plane.

May 3. Left ear (water, 8 ounces at 58 F.). Superior canal, nystagmus slow to left rotatory, 75 seconds. Horizontal canal, nystagmus slow to left horizontal, 130 seconds. Pointing error to left marked. No vertigo.

Summary: (1) Nystagmus present after stimulation of right and left labyrinth—normal in character but more easily produced with longer duration in left side. (2) Vertigo—very little, if any. (3) No pointing error in right —neither from perpendicular nor horizontal canals. Marked pointing error in left—both from perpendicular and horizontal canals.

Rotation.—Ten times in 20 seconds. Horizontal Canals—to Right:—Nystagmus—slow to right—33 seconds. Pointing—touches with right and left. Vertigo—very slight, if any. Twice repeated after interval. Nystagmus—slow to right—45 seconds; to right—30 seconds. Pointing—right hand—marked pointing error in both occasions. Pointing—left hand—marked pointing error in both occasions. Vertigo—slight on first repeating, none on second.

Vertical—to Right. Nystagmus—slow to right—48 seconds; violent fall to left. Repeated. Nystagmus slow to right, 40 seconds. Pointing—right 5 inches to right; left 7 inches to right. Violent fall to left. Vertigo—none.

Horizontal Canals—to Left.—Nystagmus—slow to left—19 seconds. Pointing—right 2¼ inches to left; left 2¼ inches to left. No vertigo. Twice repeated after interval. Horizontal—to Left.—Nystagmus—slow to left, 15 seconds; to left, 20 seconds. Pointing—right about 3 inches to left; pointing touches. Pointing—left about 2 inches to left. Vertigo—none; vertigo—slight.

Vertical—to Left.—Nystagmus—slow to left, 36 seconds. Pointing—right and left—touches. Violent fall to right, a propulsion rather than a fall. No vertigo. Repeated. Nystagmus—about 5 seconds; slow to left. Pointing—right and left touches. Violent fall to right—a propulsion to right. Vertigo—slight.

Summary.—1. Horizontal canals: (a) Nystagmus markedly diminished on stimulating right labyrinth compared with left. (b) Vertigo little on either side. (c) Pointing: very irregular; absent once on each side; when present more marked on stimulating left labyrinth and here distinctly increased on repeated pointing; not so after stimulation of right labyrinth.

2. Rotation.—Vertical canals: (a) Stimulation of left labyrinth gave normal nystagmus; marked pointing error; slight vertigo. (b) Stimulation of right labyrinth gave diminished nystagmus; no pointing error; slight vertigo. (c) On stopping, violent propulsion of upper part of body in direction opposite to previous rotation.

HISTOPATHOLOGIC FINDINGS IN PSEUDOTUMOR CEREBRI. Dr. Percival Bailey (by invitation). This paper appears in full in this issue.

DISCUSSION

Dr. G. B. Hassin said that Dr. Bailey was able to show only a few specimens illustrating the lesions in his case. Under the oil immersion a still greater wealth of changes can be made out. These changes are decidedly in the nature of acute meningo-encephalitis which, however, can be found in any other type of acute or chronic inflammation of the brain (general paresis, lethargic encephalitis, trypanosomiasis). The difference is but in the localization of the changes. To determine the proper localization, a number of sections from every part of the brain must be studied. In this case the temporoparietal lobe was found to be more involved than any other portion of the brain and it is, of course, impossible to tell whether the unequal involvement of the cortex is specific for such cases. Whatever the localization, Dr. Bailey's histopathologic and clinical studies definitely show that a meningo-encephalitis may give a clinical picture of brain tumor.

Dr. E. R. LeCount said that some years ago Pick of Prague introduced the term pseudocirrhosis of the liver, but there is no such disease of the liver and the term caused great confusion. Dr. LeCount questioned the advisability of introducing such a term as pseudotumor and thought it would be better to avoid confusion, to call it meningo-encephalitis with symptoms of tumor.

The island of cartilage in the dura interested him and he wished to know how large it was and whether there were many such islands. The dura is not supposed to make cartilage. He was also interested in the accumulation of cells about the blood vessels. He had not studied them but hoped some time to know the details of the staining according to the Schridde method of the cytoplasm of the cells that were said not to be lymphocytes. He had not been quite able to convince himself that accumulations of cells so commonly attributed to the migration of glia cells and their gathering in certain places, especially about blood vessels, were not in reality due to the passage of cells from the blood stream and collection subsequently at such places.

Dr. Peter Bassoe asked what Dr. Hassin and Dr. Bailey thought of the relationship of this sort of cephalitis to so-called lethargic encephalitis. Dr. Bassoe thought there was more hemorrhage than in lethargic encephalitis. In the latter the hemorrhages usually are recent, probably agonal but not with tissue reaction as in these specimens. In the present case there was an entirely different distribution of the lesions: little involvement of the brain stem and basal ganglions but considerable involvement of the parietal lobes. In his opinion Dr. Bailey's case corresponded more to what was previously described as influenzal encephalitis. He had not read the detailed description of so-called influenzal encephalitis, but he knew that Dr. Hassin had and wished to know whether this case was somewhat similar. He thought the case could not be included in the group of lethargic encephalitis; if so, it was the first case that had come to Dr. Bassoe's attention in which choking of the disk was present.

Dr. Julius Grinker stated that he had reported a case of encephalitis with choked disk in which the patient made an almost complete recovery. In the literature others had found choked disk.

Dr. Percival Bailey said he had tried only to establish the fact that this case, which clinically simulated a tumor of the brain, was an encephalitis. He was heartily in agreement with Dr. LeCount that the term pseudotumor cerebri had no justification. With regard to the islands of cartilage in the dura, he had tried to maintain throughout a purely objective description. They resembled more nearly cartilage than any other tissue. He left it to Dr. Hassin to answer the more technical pathologic questions but was aware that Tschistowitsch, working in Ziegler's laboratory, had demonstrated as early as 1898 active division of the glia cells in the repair of aseptic wounds of the brain.

Dr. G. B. Hassin said that Dr. Bailey, in his case, made a special study of the midbrain and substantia nigra, the regions that are always affected in lethargic encephalitis. In his case they were found practically normal. The hemorrhages in his case were small, microscopic and rare, and always accompanied by marked reactive phenomena. Such a type of hemorrhagic phenomena does not occur in lethargic encephalitis. On the other hand, in cases described as influenzal encephalitis, hemorrhages are the prominent feature without reactive phenomena and other changes, like perivascular infiltrations, parenchymatous lesions, etc. Such cases, commonly known as hemorrhagic encephalitis, run a rapid course while in Bailey's case the duration was three months. An attempt was made to explain the hemorrhages in the case under discussion on the basis of the operation, but this was performed three months before the patient's death and the hemorrhages were not confined to the place of operation, but to remote regions; like the optic thalamus and pons. In short, the hemorrhages like the rest of the pathologic findings were not traumatic, but of some infectious inflammatory origin. It somewhat differs from other types of encephalitis (lethargic, general paresis) by the localization of the pathologic changes and the presence of preagonal hemorrhages. It remains to be seen whether other cases of so-called pseudotumor give findings similar to those in Bailey's case. The numerous small nuclei scattered in the brain tissue are glia cells, not lymphocytes. Dr. LeCount doubts whether they can, with absolute certainty, be differentiated from lymphocytes that emigrated from the blood vessels. Without proper staining methods, he thinks, a differentiation cannot be made. The differentiation between these two types of cells, lymphocytes and glia nuclei, is not made from their staining qualities, which are practically alike, but on the basis of their embryologic properties. The lymphocytes are mesodermal elements, the glia cells ectodermal. These elements do not mix with each other, but occupy territories strictly assigned to them. Thus, the lymphocytes are confined to the vascular system and the perivascular spaces, while the glia cells are confined to the brain tissue outside the blood vessels. This teaching, promulgated by Nissl and his school, proved to be correct and is universally accepted by neuropathologists.

Dr. Ralph C. Hamill said that in a specimen showing diffuse melanosis of the cord and basal portion of the brain, large cells considered to be glia cells because of the presence of glia fibers were seen to contain many fine granules. These granules, because of their staining reaction and because of other evidences of hemorrhages, were believed to be of hematogenous origin.

Dr. E. R. LeCount said the granules he had in mind were those of the cell bodies or cytoplasm. Lymphocytes had been studied according to their granulae reaction to stains and classified rather extensively by those working in general pathology. He did not know whether this had been done in connection with the brain and until it has, one should not accept a dictum as to their origin—and possibly not then.

THE PRESENT STATUS OF THE ABDERHALDEN REACTION.

Dr. Clarence A. Neymann.

Perhaps no serologic reaction, the Wassermann test of syphilis excepted, has caused such wide discussion as the Abderhalden reaction. Authors closely associated with Abderhalden reported specific results with the test. Other investigators, among them some of the best trained, reported nonspecific reactions in pregnancy and other conditions. Fauser was the first to apply the test in psychiatry and reported positive reactions for testis, ovary and other glands of internal secretion in dementia praecox. Van Slyke separated the method from the questionable factor of the permeability of the dialization tubes. A dialization tube, even though just tested, might become worthless during a test and permit the passage of protein, thus nullifying the value of the Ninhydrin reaction. The quantitative aminonitrogen method for blood serum—which gives the amount of the aminonitrogen present in the serum, before and after incubation of such a serum with the protein against which protective ferments are supposed to exist—does not confirm the original premises of Abderhalden. Thus, notwithstanding the later protest of Abderhalden himself, we cannot consider his theory or reaction as established.

Notwithstanding these facts, the Abderhalden test was employed for testing about one hundred different serums. The work was done by Dr. Frances L. Dunham and myself in the Phipps Clinic, Johns Hopkins Hospital. We have not heretofore published the results as they seemed to approximate those obtained by many other investigators. The dialization method was used with the technic recommended by Abderhalden in his "Abwherferment," fourth edition. The serums of ninety-seven patients were examined. Of these, forty-four were classed by us as of the schizophrenic type, thirty-nine suffered from various mental disorders, eight were normal pregnancies and six normal persons, male and female. Our material was quite different from that encountered in state hospitals. Most of the patients were in the earliest stages of their psychoses.

As is well known, all shades of blue can be obtained with the ninhydrin reaction. For our purposes, which were essentially clinical, the only thing that concerned us was whether a reaction was positive or negative. Whenever the dializate of the control tube, as happens with some serums, was slightly colored, and the dializate of the tube incubated with substrate was definitely blue, the reaction was considered positive.

Thirty-two of the forty-four serums of the patients of the schizophrenic reaction type were positive. Twelve were negative to sex glands. This corresponds roughly to 73 and 27 per cent. There was, however, a certain "overlapping" of the reaction; that is to say, of the twenty-five positive cases also tested with placenta as a control, five gave faulty positive reactions with this substrate. There was, therefore, a definite tendency for the serums of patients of the schizophrenic reaction type to give a positive ninhydrin reaction with sex glands. The results obtained with the serums of thirty-nine persons not of the schizophrenic reaction type, thirty of which gave negative, nine positive reactions with sex glands, corresponds to about 77 and 23 per cent., respectively. This is about the inverse ratio found in the first group, and would seem to show a definite and undeniable difference in the biochemical make-up of the serum of most schizophrenic when compared with the serum of other mental patients. There seems to be no doubt that the serums of schizophrenic patients are less stable when incubated and dialized with various organs and glands, than the serums of other patients. This instability is more easily brought about by sex gland tissues than other organic tissues. We believe that there must be some factor as yet unknown on account of which the serum of such patients is especially prone to dialize.

Recently Dr. Retinger has striven to overcome the many technical difficulties by using celloiden sacks for dialization and by allowing the serum to react with several sets of substrata. In his 1918 publication of twenty-five cases, four of which were controlled by necropsy examination and two by operation, we can see only those points of interest which we have seen throughout the development of the test—striking results balanced by peculiarities which at times were just as striking.

The most recent serums sent Dr. Retinger for examination have been interesting. We have only sent serums of such cases as could be absolutely concontrolled. One negative reaction was reported as a large parietal tumor through necropsy by Dr. LeCount. The other serum from a man with manifest endocrine disturbances, cryptorchism, or possibly Froelich's syndrome, was reported as giving a reaction with pineal gland and pancreas. All other glands of internal secretion gave negative reactions. In our opinion, many of these glands were affected. Thus, even with excellent technic and extreme care, unaccountable results can be obtained. We must, therefore, conclude that the Abderhalden reaction is interesting, may at times point in the right direction, but is thus far valueless for diagnostic purposes in psychiatry.

DISCUSSION

Dr. J. M. Retinger: The poor results obtained by most of the later investigators of the Abderhalden test are due to faults of technic and lack of experience. If we speak only about its application to the diagnosis of pregnancy, we find the following sources of error:

1. Unreliable Dializers: The judging of the original parchment sacks is extremely difficult.

2. Toluol: Toluol used as antiseptic contains many acid impurities which interfere with the digestion and the ninhydrin reaction.

3. The Substrate: Not all placentae prepared in exactly the same way are equally sensitive; some are indigestible and some are oversensitive, both probably due to minute pathologic changes.

4. Interpretation of Results: Placenta is to a large extent vascular tissue. If the patient suffers from any disease of the blood vessels we would naturally obtain a positive reaction, which cannot be interpreted as digestion of the placental specific protein.

5. Some serums have enough amino-acids in solution to give blue color with ninhydrin, a fact that is rarely taken into consideration.

The presence of specific ferments and their specificity was proved by Abderhalden by these tests: Ninhydrin reaction, polariscope, refractometer, the Van Slyke amino-acid N method, and colorimetric method, using stained proteins as substrata.

The presence of specific ferments and their specificity was proved by Hirsch by the interferometer, by Kantorowitch by the biuret test and by Kottermann by a colorimetric estimation of iron split off from ferrated substrata.

In my own experiments with the ninhydrin test I have obtained perfect specificity. These ferments appear to be even more specific than Abderhalden himself asserts. The specificity is so great that histologically identical parts of the brain could be differentiated sharply, provided their function was different. This observation was substantiated in a series of over one hundred cases of gross brain lesions when the lesion could be localized properly with this technic. Naturally this technic cannot be considered as based on a wrong theory, accidental results or a constantly repeated error, because only four cases of the series were localized wrongly, and because these results were obtained after almost six years of experience in this work.

Dr. Neymann (closing) said, regarding the question of substrata that he, while working in Heidelberg, took up this question of overlapping and found reactions with carcinomatous substrata and pregnant serums. After preparing tissues he sent these to Abderhalden and had them approved by him and used these tissues with the precautions recommended by Dr. Retinger. The same overlapping was seen. The tissues used in the test with dementia praecox serums were prepared with the greatest care. In order to make sure that they did not consist solely of connective tissue they were stained and examined under the microscope. He thought Van Slyke had done this much better than he, and even then the theory did not hold good.

Book Review

THE PROBLEM OF THE NERVOUS CHILD. By Elida Evans, Introduction by C. G. Jung; Dodd, Mead & Co., 1920, pp. 299.

It should be fairly stated at the outset in a professional review of this book that it disclaims being a presentation for professional people and that it deals very little with nervous children in the medical sense. The gist of the situation seems to be that Mrs. Evans, through her experience "in the difficult and toilsome treatment and education of nervous children," has developed a fund of practical wisdom that has led her to write this book "to aid those parents who in the training or education of their children have arrived at the point where the methods already used have proved inadequate."

There are various parts of the book that one heartily wishes might be read and reread by parents—the parents that one comes to know when studying difficult children—but it seems much less certain that the book as a whole can be considered a desirable dose to prescribe for any given parents. There seems to be danger in the many indiscriminating doubts and fears that the reading of the book, if it is taken seriously, will engender in the conscientious concerning their own behavior as it affects their children. Pitfalls for the young mind appear on every hand and how to cultivate a terrain free from serious danger during the developmental period seems altogether an uncertain task. Indeed, it seems difficult to explain how so many children get through without obvious injury when all the paths to disaster that Mrs. Evans enumerates are considered. These poor, foolish, silly, selfish, despotic, domineering, busybody parents with their lack of reasoning power and their "past of ignorance," as Mrs. Evans depicts them, undoubtedly trammel the growth of their children's lives, and psychanalysis of the parent would often be the best prerequisite of treatment of the child. The main point set forth is that the behavior and attitude of parents hamper the finest growth of children rather than that out-and-out nervous disorders are caused thereby.

It is going quite a long distance further to intimate that dementia praecox may be caused by the untruthfulness of a mother or that the fear of punishment causes epilepsy. As a matter of fact, however, many of the most common nervous states of children are not considered at all in this work, and many of the cases cited seem to be merely those of difficulty in social reactions or adjustments just as we should expect, with these backgrounds of unwise parental conduct that she cites.

The reader is offered occasional easy sermonizing on various subjects—bachelors in clubs, conditions in Russia, labor troubles—that seem hardly indicated by the title. In many places the interpretations are clearly of the cocksure and seemingly far-fetched sort that users of symbolism and dream material indulge in.

However, nothing quite so sticks in the reviewer's crop as the social or economic implications of this work. Difficult children—and if possible their parents—are to be studied and treated by the regular psychanalytic method. Of course we read, then, of the many weeks and months and even several years over which the author's cases have been analyzed. But what earthly chance is there of doing this except for the few favored (or unfortunate) persons who have wealth? And if good results are so delayed and are not always obtained (Mrs. Evans fails to give final outcomes in many instances), it is an open question whether there must not be vastly more direct routes to self control or sublimation, such as punishment, social or physical and even corporal, or through such exigencies as economic necessity for early meeting the world on its own terms by working for a living or through strong moral or religious influences. How otherwise are people working themselves through their difficulties—is it only the well-to-do that do have these sterner chances for conquering themselves or receiving simpler aid?

Indeed, is it not a fact that the great part of the whims and vagaries of behavior, which in this book are apparently offered as the symptoms of "The Nervous Child," are only possible in circumstances of some affluence? There can be little doubt that a highly protected environment gives proportionately more opportunity for unfortunate reactions of any inner urge (libido). The world of her patients, we glean from Mrs. Evans' descriptions, is one of rich velvets and many toys and pleasures for the children, fine jewels or big business interests for the parents. Not that we would maintain that poverty gives freedom from untoward effects of mental conflict—the reviewer has published many cases showing the opposite. But the point is that psychanalysts have too little faced the fact that certain social and economic circumstances largely condition the ailments of their clientele as well as create the possibilities of psychanalytic treatment. The frank statement of some that they will do nothing for those who come from a "lower" social level or who cannot pay well may be in recognition of the immediate cardinal facts, but that makes it possible to draw some interesting practical deductions from two angles.

To form a book best adapted for parents on parental influences and correlated possibilities for good and evil, it seems to the reviewer that a culling process might well be undertaken and the many good paragraphs be separated from the mere fanciful and less surely founded features of the text. The latter will draw the reader's attention, perhaps somewhat morbidly, but, after all, these passages may so embody the author's own credo that she would be unwilling to drop them.

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WOUNDS OF THE HEAD AND COMPENSATION LAWS *

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INJURIES AND COMPENSATION

There are now forty-two states and two territories in this country that have compensation laws. It is estimated, approximately, that two and a half million accidents occur yearly to workmen (four-fifths being trivial) and that $120,000,000 are spent on them. In spite of all preventive efforts the number of accidents is said not to be decreasing.

There are about 2,500,000 workmen in New York state subject to the compensation laws (about 80 per cent. of all workmen). There were 288,441 accidents reported in 1919. About 20 per cent., or 57,550 were compensated, and the total compensation in 1916 was $12,803,000, about $12,000,000 being in cash benefits and about $180,000 for medical services. In New York, if a workman is injured enough to lay off for two weeks, he receives compensation, namely, medical attention and $20 per week for two months. If at the end of two months he is not well, he goes to the State Industrial Commission, which decides whether his injury is a total or partial, complete or temporary disability: (1) permanent, partial and (2) temporary total disability, are the two main groups, for which compensation is given.

Large numbers of applicants for further relief appear at the commission office, about two months after the accident. They have meanwhile been under the care of insurance and other physicians, but are still disabled—or think they are.

About 15 per cent. of these applicants complain of nervous symptoms and a great many of them have received wounds of the head or have fallen. These accidents have been followed by symptoms of brain concussion, brain contusion, fracture of the skull, and other

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* Read at the Forty-Sixth Annual Meeting of the American Neurological Association, New York, June, 1920.

conditions. These patients are referred to various neurologists and their stories are no doubt familiar. Dr. Michael Osnato has made a study and analysis of 200 such cases, and has given statistics of the age, social condition and symptomatology, classifying them in two groups—(1) traumatic neuroses, (2) traumatic hysterias.

The frequency of wounds of the head which are not fatal and which do not cause definite crushing or hemorrhage of the brain is great, but there is yet no satisfactory explanation of the symptoms or sure information about the prognosis. A study of the natural history of these wounds should be made. I have seen and examined many cases of this kind in the last few years, and have gone over a number of my earlier cases of traumatisms of the head in which litigation was sometimes involved and sometimes not.

I am not analyzing these cases, since Dr. Osnato has done it carefully and well. My study has been made rather to inquire into the nature of the injury and what can best be done for these cases by the profession and the state.

It seems to be the opinion that the present compensation laws are doing good on the whole, but are imperfect and in many states ineffective. In so far as they relate to the treatment of wounds of the head, I think that improvements are needed and can be made and I think that those officially connected with compensation work have an open mind on the subject.

HEAD WOUND SYNDROME

The syndrome itself of nonfatal, nondestructive wounds of the head is quite a definite one. From whatever source gathered the story is about the same. In Dr. Bailey's book on injuries of the nervous system, in a description of traumatic neurosis published years ago by myself, (Hamilton's Legal Medicine), in Chatelin and Demartel's "Wounds of the Skull and Brain," the lists of subjective symptoms given have about the same items.

The patients from the compensation commission who come with bruised heads tell stories so startlingly similar that it seemed to me finally that there must be an underground school for the education of those who have been hit on the head and desire permanent total disability compensation.

This syndrome of wounds of the head as described by Bailey[1] is: headache, vertigo, irritability, sleep disturbances, asthenia and memory defect.

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[1] Bailey: Injuries, p. 141.

The symptom group given by Chatelin and Demartel,[2] and "found in cases with and without sign of objective trouble" is: headache, vertigo, insomnia, changes in character, memory defects, vasomotor phenomena, sweats, palpitations and flushings. My own records of compensation cases give about the same list: headache, vertigo, insomnia, irritability, anxiety, depression, memory defects, fatigability; tinnitus, partial deafness and loss of weight. Thus the wound of the head syndrome is a well established one. It follows wounds of the head in war time, in civil life with litigation and compensation and without either. It is a picture of a general traumatic neurosis or psychoneurosis with certain local head symptoms, which have a possible legitimate basis.

NATURE OF SYMPTOMS

The experience of surgeons shows that when the brain has been badly contused and the skull injured, headache and attacks of something like vertigo or mental confusion occur. In bad wounds and contusions the headache is intense at times and even calls for surgical relief.

It is probable that in all severe direct blows on the head there is some contusion of the brain, but in most cases it is trifling and it is generally stated that the wound of the head syndrome disappears in a few months.

I classify the patients as follows:

1. In about 10 per cent. of compensation cases with head wounds the patient has the main features of this syndrome and is just made uncomfortable by it. One cannot say he has any neurosis or psychosis in the sense that there is any general constitutional disturbance of the nervous centers.

2. In a larger percentage, that is, in more than half the cases, in addition to headache, vertigo and insomnia, the patient is anxious, worried, nervous and depressed and cannot concentrate or work. He has never been ill or injured before. He doesn't understand why he is not cured by the doctor or the insurance companies and he asserts that he wants to feel well and get back to work. He becomes selfcentered, exaggerates, refuses to attempt to work or to do anything but wait and get some new treatment that will make him well.

3 and 4. In another large percentage of patients, contributed largely from certain races, from the beginning or after an incubation and the sympathetic suggestions of friends, the patients develop a definite attitude of exaggeration or become plain malingerers. They see ahead

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[2] Chatelin and Demartel: Wounds of the Head, p. 38.

a lump sum of several hundred dollars. Various neurotic and hysterical symptoms develop in connection with these attitudes of mind.

There are some definite facts about these cases:

1. The patients have been distinctly and seriously injured, in most cases knocked unconscious for periods varying from a few minutes to a few days.

2. They have developed a definite and, we may say, normal syndrome of head wounds.

3. They have no specific evidence of any localized brain injury—no evidence of any central organic nervous disease, except that we may suppose there has been some slight brain bruise or local meningeal reaction or a labyrinthine injury.

4. They are receiving weekly compensation and many expect to be pronounced more or less disabled and receive some lump sum which is rarely more than a few hundred dollars.

5. When their case is settled, the symptoms do not always disappear; and, after their final award is spent, in some cases they come back with the same or more symptoms, and try to get more money.

6. In similar wounds of the head in nonlitigated cases the symptoms sometimes continue and may even grow worse.

I think every one has had cases of these kinds under observation. How are we to explain the symptoms? It is not enough to classify them into traumatic neuroses and traumatic psychoneuroses in accordance with the amount of neurotic or psychic element in the cases. I place them in the four categories just referred to.

The psychology is, in most cases, simple and does not involve the subconscious very much. Indeed most of the patients are dull, extremely ignorant, and do not appear to have any organized subconsciousness, much less a directing complex or the mechanisms of a subconscious defense. They are uncomfortable, suffering, apprehensive, hopeful of getting a sum of money, or they are perhaps enjoying life without work for the first time and receiving money weekly like the idle rich, while the wife works harder at home or goes out and earns more money. They are, in fine, under the various conscious drives of the instinct for food and an easy life, the instinct for native home, the fear of death and disability, the fear of the future and uncertainty of settlement. In many cases we may suppose that on the basis of the brain concussion injury together with the emotional reaction associated with it and an expectation of relief and reward, disassociations occur, the synapses break down and so-called hysterical symptoms appear. Then they are said to have a traumatic psychoneurosis.

My feelings toward most of the patients, however, is that to call them victims of neuroses or psychoses is hardly worthy of our science.

They have just traumatic conduct disorders. They suffer hardly even from conduct disorders, but from what are of the nature of antisocial reactions to a painful and incapacitating injury.

Aside from or with the subjective symptoms which I have described there occurs a percentage of real injuries, such as fracture of the bones, severe strains of the neck and back muscles or a definite bruise of Broca's convolution with aphasia. A progressive muscular atrophy appeared in one case. In elderly persons, over 50 or 60 years of age, a cerebral and general arterial sclerosis is found and senility seems to be accelerated.

Morbid fears which are associated with vertigo naturally develop. A man who has been working on high buildings, a painter who has to climb ladders, does not want to go back to his calling and unless he is quite young he probably never can do so. But these are physiologic fears, at least they do not necessarily involve any complexes or subconscious defense reactions. A good many patients develop "habit pains" which may be explained as "conditional reflexes."

TREATMENT

Most cases could be relieved early and entirely if properly handled; this is the important issue I would emphasize.

If after an injury the state refused all compensation, all final awards for disability, probably there would be 50 per cent. less who would continue to have the wound of the head syndrome; but there would still be many left. The state cannot now undertake this change, but it could do something. Now it spends $12,000,000 on compensation and $180,000 on medical care. It should spend its money on the immediate and skilful care of the injured man. He should be early diagnosticated, instructed and intensively treated and reconstructed, and he should be emphatically told that his functional troubles will receive no recognition as permanent disability.

CONCLUSIONS

My conclusions are that there should be such a modification of compensation laws, that the patient receive early expert advice, not only surgical but neurologic; that treatment and reeducational and reconstructive measures be early and actively employed; that the closest possible study be made early to determine the existence of local brain and cord injuries. Perhaps some day objective tests may be discovered that will detect exaggeration and malingering.

Until then we must rely on experience and acuteness of observations and on the intensively detailed history of the case.

CHOREO-ATHETOID AND CHOREOPSYCHOTIC SYNDROMES AS CLINICAL TYPES OR SEQUELAE OF EPIDEMIC ENCEPHALITIS *

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Epidemic encephalitis, also known as lethargic encephalitis, nona, infectious ophthalmoplegia and by various other designations, has only recently sprung into prominence but by reason of its rapid spread over the greater part of both hemispheres in less than two years, it has engrossed to an unparalleled degree the attention of scientists throughout the world. Although von Economo's first description of this disease only dates back to the spring of 1917 the amount of literature concerning it has already reached incredible proportions, a fact which has served promptly to vulgarize the symptomatology and diagnostic features of this newly isolated affection and also to provide us with a preliminary knowledge of the character and distribution of the underlying histologic changes.

It seems well established that the lesions involve with especial predilection the tegmentum of the pontopeduncular region and the basal ganglions, and give rise clinically, in the majority of cases, to a fairly distinctive symptomatic picture characterized essentially by fever, progressive lethargy and asthenia, ocular and facial palsies, more or less generalized rigidity or passivity and occasional myoclonic twitchings. The frequent occurrence, however, of quite dissimilar though almost equally typical syndromes affords presumptive evidence that in this disease the histologic changes may be disseminated through the entire nervous system with focal predominance at any given level. It probably will be shown in time that, much in the same fashion as in poliomyelitis, the changes in epidemic encephalitis are generalized in the great majority of cases and that what is now regarded as the classic clinical physiognomy of the disease simply depends on the commonly overwhelming preponderance of the lesions in the periventricular gray matter compassed by the striatothalamotegmental region. This is indeed the favorite localization of the morbid process in epidemic encephalitis and its clinical expression has been so abundantly studied and widely depicted that it can hardly escape detection. It is just as important to recognize the more unusual or atypical forms of the disease, though, be it stated at once, they are atypical only

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by contrast with the more familiar clinical picture sketched in the foregoing. In reality they represent various aberrant or level types in which the salient clinical features are so strikingly foreign to the usual cardinal grouping that diagnosis is frequently jeopardized. Many clinical types of epidemic encephalitis have already been encountered and described and the constant report in the literature of additional syndromes bids fair to extend almost indefinitely the possibility of revised classification of the numerous forms or syndromes to which the remarkably protean symptomatology of the disease may give rise.

Several authors have already hazarded a practical tabulation of the various clinical types of epidemic encephalitis, and to some such a classification at this time may, perhaps, seem somewhat premature, but when one fully realizes the not infrequent difficulties in differential diagnosis which arise in connection with certain forms resembling alcoholic delirium or general paresis, cerebral tumor or cerebral abscess, acute meningitis or poliomyelitis, vascular lesions of the cerebellum, etc., any attempt at early classification will be conceded to be both a judicious and laudable effort. Furthermore, it is no idle task to utilize the valuable and suggestive data furnished by the variable clinical aspects of epidemic encephalitis in a legitimate endeavor to elucidate many obscure problems of cerebral physiology and localization. There can be no doubt that the intensive study, both anatomic and clinical, of this newly identified cerebrospinal affection promises to shed much light on the pathogenesis of several hitherto baffling neurologic disorders such as paralysis agitans, choreiform and athetoid states, endocrinal dysfunctions and even certain psychoses and psychoneuroses.

The various clinical forms of epidemic encephalitis thus far signalized include the following types: lethargic, cataleptic, polioencephalitic, anterior and posterior poliomyelitis, Parkinsonian, acute psychotic, epileptomaniacal, meningitic, meningoradicular, polyneuritic, cerebellar, hemiplegic, diplegic, monoplegic, as well as a form occurring in infants and called for this reason encephalitis neonatorum. In reality there are still other types, and by the time the chapter devoted to this disease has been fully written we probably will find that it embraces clinical syndromes that reproduce with surprising precision the outstanding features of many of the known acute and subacute diseases of the nervous system.

A point which cannot be too strongly emphasized is that aside from a classification based on predominating clinical features and intended to establish the existence of level types, a classification based on the course or evolution of the disease must likewise receive due consideration. Some authors regard the disease as having essentially an acute onset, while others describe the onset as subacute or insidious. The fact seems to be that both modes of onset are about equally common. The duration of the disease is extremely variable, stated by most observers as being approximately eight weeks in the majority of cases. A hyperacute or fulminating course is extremely rare, yet I have seen two undoubted cases of epidemic encephalitis terminate fatally in less than a week with acute bulbar paralysis. It is becoming more and more evident likewise that a very prolonged or chronic course extending over several months is far from uncommon and this fact is of the greatest importance in explaining the frequent relapses or the recrudescence of certain symptoms observed in patients who, because of the relatively mild and purely evanescent character of their initial disturbances, have been prematurely discharged from hospital service or allowed to resume work. This same factor of chronicity affords an explanation of certain unusual syndromes which soon follow the complete disappearance of a few abortive manifestations of the usual type of the disease and even continue to develop in the pseudoconvalescent stage until a fairly well defined parkinsonian or choreoathetoid syndrome is materialized. Under these circumstances we are apt to interpret such developments as sequelae of the disease, while in reality they are simply the expression of slowly evolving degenerative changes in the cellular complexes of the basal ganglions engendered by a prolonged or even stealthily increasing inadequacy of the vascular supply. Finally, the occurrence of an abortive form of epidemic encephalitis is of the greatest importance and it is more than likely that the number of cases belonging under this head fully equals or even exceeds that of frankly developed cases. Here again the true origin of transitory or substantially recoverable though frequently severe types of psychoneurosis and mental disorder may remain totally unsuspected.

I have seen a total of about sixty cases in which the diagnosis of epidemic encephalitis could be postulated with relative certainty. A factor on which I have relied to a considerable extent is the presence of changes in the cerebrospinal fluid, more particularly a cell count varying from 10 to 200 per cubic millimeter associated with a definitely positive globulin reaction and with otherwise negative findings, i. e., a negative Wassermann reaction and absence of micro-organisms. Many observers have reported series of cases in which the spinal fluid was almost universally negative. Such has not been my experience and in the great majority of cases the fluid was definitely pathologic. I believe that the laboratory findings depend very largely on the stage of the disease and that the conditions are analogous to those encountered in poliomyelitis, namely, the frequent disappearance of definite changes in the spinal fluid after two or three weeks. Aside from the fact that there are mild or abortive cases with minimum modifications of the spinal fluid, there can be no doubt that many cases of epidemic encephalitis come under proper observation long after the truly initial phase of the disease. This combination of factors may account in part at least for the apparent discrepancies in the report of different clinicians.

Included in the series of sixty cases which have come under my observation were instances which correspond to most of the types thus far described in connection with this disease. Though pains in the extremities and cerebellar manifestations were not particularly uncommon, I have not seen frankly polyneuritic, herpetic or cerebellar types. I have seen, however, eight or nine cases which deserve to be designated as choreopsychotic and choreo-athetoid types of epidemic encephalitis because such manifestations represented in these cases the persistently predominating features of the clinical picture. Inasmuch as these clinical forms do not seem to have attracted much attention, at least so far as one may judge from the relatively meager reference to them in the literature, it has seemed useful to publish a few case histories which serve to illustrate these rather unusual types of epidemic encephalitis. This does not mean, of course, that either choreiform twitchings or psychotic manifestations have not been frequently recorded in connection with the disease. On the contrary, in the very earliest accounts of lethargic encephalitis emanating from Great Britain and France, more particularly in the report of the special British government board[1] and in the initial communications of Netter,[2] the occurrence of choreiform or analogous movements in some cases was emphasized. In this country, Bassoe[3] was apparently the first to note these same phenomena which were present in four of the twelve cases originally reported by him. The choreiform twitchings involved the extremities and in two of the cases were confined to the left leg.

In all of these cases, however, the choreiform jerkings simply represented one of the many striking features in the clinical grouping. The same is true of another hyperkinetic phenomenon more recently identified and consisting of rapid rhythmic myoclonic twitchings in the abdominal wall, the face and the extremities, resembling the shock-like contractions induced by electrical stimulation. This myoclonus, which was described more particularly by Sicard and Kudelski,[4] Sabatini,[5] and Reilly,[6] is quite commonly observed, now that attention has been directed to it, in cases of epidemic encephalitis irrespective of the clinical type under observation. In some instances, however, it constitutes such a predominating feature that Sicard and Kudelski were led to isolate a special form to which they have given the name of "encéphalite aigüe myoclonique." In this type of the disease the myoclonic twitchings are described as brief, rapid and explosive, of distinctly rhythmic character and involving the musculature of the limbs, face and diaphragm; they are preceded by lancinating pains, headache and moderate fever and persist throughout the delirious and stuporous manifestations which eventually develop and usually prove fatal. The ordinary symptoms of epidemic encephalitis, such as somnolence and ocular palsies, are wanting. Sabatini likewise has laid great stress on the frequent occurrence of myoclonic phenomena and has given an excellent description of the symptom which fully tallies with that of the preceding authors. Sabatini insists on the fact that in some cases the jerkings simulated actual chorea. It should be stated, however, that there are very essential differences between chorea and rhythmic myoclonus, and that in reporting the present series of cases I have in mind truly choreiform or choreo-athetoid manifestations and not regular rhythmic spasms.

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[1] Report on an Inquiry Into an Obscure Disease, Encephalitis Lethargica, Local Government Board Reports on Public Health and Medical Subjects, N. S. 121, London, H. M. Stationery Office.

[2] Netter: L'encéphalite léthargiéque, Paris méd. **8**:8 (Aug. 3) 1918.

[3] Bassoe, Peter: Epidemic Encephalitis (Nona), J. A. M. A. **72**:971 (April 5) 1919.

As regards psychic disturbances in epidemic encephalitis, their common occurrence has been signalized from the very start and many authors have called attention more particularly to the nocturnal agitation with delirious states of hallucinatory or even delusional character. Here again in the majority of cases the psychic disorder has been merely one symptom of an otherwise well characterized instance of the usual type of epidemic encephalitis. In some cases, however, the mental confusion, agitation, hallucinosis, etc., have been so markedly predominant as to quite overshadow all the other features of the disease and to legitimate the description of an "acute psychotic type." Tilney [7] has thus designated this form of the disease. Rather similar observations were published by House [8] and others, and as far as

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4. Sicard and Kudelski: L'encéphalite aigüe myoclonique, Bull. de la Soc. méd. d. hôp. **44**:94 (Jan. 23) 1920.

5. Sabatini: Lethargic Encephalitis: Symptoms and Course, Policlinico **27**:97 (Jan. 26) 1920; abstr., J. A. M. A. **74**:1057 (April 10) 1920.

6. Reilly, Thomas F.: Hitherto Undescribed Sign in Diagnosis of Lethargic Encephalitis, J. A. M. A. **74**:735 (Mrach 13) 1920.

7. Tilney: Epidemic Encephalitis, A Preliminary Consideration of Some of Its Prominent Clinical and Pathological Manifestations, Neurol. Bull. **2**:106 (March) 1919.

8. House, W.: Epidemic (Lethargic) Encephalitis, J. A. M. A. **74**:372 (Feb. 7) 1920.

the psychic disturbances are concerned, the cases reported by these authors were remarkably similar to those described in the present article.

The combination of marked and persistent psychic disturbances with incessant choreiform jerkings as the outstanding features of a syndrome produced by epidemic encephalitis appears to be far more uncommon, and typical cases of this sort were not encountered in the literature until very recently when a further contribution to this subject by Bassoe[9] came to my notice. Two of the cases reported by this author somewhat resemble those which have come under my observation and which are herein designated as the choreopsychotic type. This form of the disease is, I believe, far more common than is generally supposed.

Typical choreic and choreo-athetoid or frankly athetoid syndromes and other related spasmodic states occasioned by epidemic encephalitis have been particularly infrequent and have been encountered mainly as tardy or residual developments. Cases belonging under this heading have been reported by Pierre Marie and Mlle. G. Lévy,[10] Souques, Lesne[11] and von Economo.[12] Marie and Lévy published the case histories of seven out of thirteen cases of this kind. In two of the cases the hyperkinetic phenomena were essentially choreic in type, while in four others the movements consisted of rather coarse displacements of great amplitude in the leg or arm, or in both extremities on the same side, accompanied by oblique flexion or extension of the head. These spasmodic manifestations were not rapid and jerky but rather slow and relatively rhythmic and coordinate. The authors feel that they cannot assimilate these disorders to any of the actually recognized categories of involuntary spasmodic phenomena. Lesné's article deals essentially with the development of choreiform movements after the acute stage of lethargic encephalitis or as a sequel of influenza. Personal observations are recorded and the earlier experiences of Netter, Marie and Souques with analogous conditions are summarized. Von Economo has reported an interesting case of lethargic encephalitis remarkable for its chronicity, in which the condition had an acute onset and exhibited a number of remissions until more serious involvements eventually appeared and proved fatal almost two years after the initial symptoms. The patient gradually developed the syndrome of pseudobulbar paralysis associated with typical athetoid manifestations. It was characterized by dysphagia, dysarthria, dysphonia, paresis of the lingual and palatal musculature, generalized rigidity, spasmodic laughter and choreo-athetoid movements in the face as well as in the upper and lower extremities. The transitory improvement concerned more especially the cranial nerve functions whereas the athetoid disorders steadily increased in severity. At times, however, all the symptoms seemed to recede only to return in more alarming form.

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[9] Bassoe, Peter: The Delirious and the Meningoradicular Types of Epidemic Encephalitis, J. A. M. A. **74**:1009 (April 10) 1920.

[10] Marie, Pierre, and Lévy, Mlle. G.: Plusieurs cas de mouvements involontaires d'aspect particulier apparus après un épisode fébrile grippal et, pour certains, après des signes d'encéphalite léthargique, Rev. neurol. **35**:300, 511, 1919.

[11] Lesné: Mouvements choréiformes observés à la suite de l'encéphalite léthargique ou de l'influenza, Bull. Soc. méd. d. hôp., Nov. 29, 1918.

[12] Von Economo: Encephalitis lethargica chronica, München. med. Wchnschr. **66**:1311 (Nov. 14) 1919.

CHOREO-ATHETOID TYPE

Case 1.—C. W., aged 57, married, a traveling salesman, was seen by me May 5, 1919, about three months after the onset of his disturbances. Reliable data were obtained from his family physician regarding the initial phase of his disease.

History.—His family and personal history yielded nothing of significance for the interpretation of his actual nervous disorder. Both the patient and his physician were quite sure that he had not had influenza and that the first episode in his illness was the appearance of a large and annoying carbuncle on the back of the neck during the first days of February, 1919. Shortly thereafter the patient became feverish and drowsy, complained of blurred vision and diplopia and developed bilateral facial paralysis. There was neither vertigo nor vomiting, but pain of considerable severity was felt over a circumscribed area in the right parietal region which, judging from the patient's own description, had all the characters of "clavus hystericus." He soon showed evidence of some weakness in the extremities on the left side and exhibited restless movements in the trunk and extremities. He was seen at that time by a consultant and lumbar puncture was performed. The spinal fluid is said to have been entirely negative. The blood and urine were likewise negative. The patient had a slow and tedious convalescence with unsatisfactory retrogression of some of his disturbances and decided aggravation of others.

Examination.—When seen by me for the first time, May 5, 1919, he complained of recurrent facial weakness, disordered speech and uncontrollable movements of his face and extremities. Neurologic examination showed that his contentions were justified. Whether standing or sitting some part of the body was almost constantly in motion, the face and left lower extremity assuming the major share of the otherwise general instability. In the sitting posture the trunk exhibited rather slight oscillations either forward and backward or laterally, the head was slowly rotated from side to side and at the same time thrown slightly backward, the eyes were held semiclosed by the constant wrinkling of the forehead and orbiculopalpebral spasm, the expression being typically that of one attempting to distinguish a distant object in the full glare of the sun, with perhaps the added appearance of acute anxiety and distress. The angle of the mouth on either side was likewise occasionally retracted and the lips curled or twisted in various ways. The hands and forearms were now and then the seat of restless fidgety movements of pronation together with clutching movements of the fingers, but the unrest manifested in the upper extremities was minimal when compared with that observed in the lower extremities. There was ceaseless shuffling of both feet and more particularly of the left foot which was constantly displaced forward and backward or laterally, or else only the heel or forepart of the foot was alternately moved inward and outward. Occasionally the knee was repeatedly adducted and abducted. His gait showed slight dragging of the left lower extremity with uneven stepping. There was no gross paralysis in the limbs, he could execute all the ordinary movements usually tested but his motor power was generally subnormal with more appreciable loss on the left side. His grasp, registered with Mathieu's dynamometer, gave 70 on the right side and 50 on the left. The tendon reflexes were all hyperactive and distinctly more so on the left side, but neither ankle clonus, Babinski nor Oppenheim phenomena could be elicited. The abdominal and cremasteric reflexes were not obtained. The left facial nerve was decidedly paretic in its lower branch, faintly so in its upper branch. Ocular excursion was excellent in all directions save as regards convergence which was practically nil. The pupils were small, perfectly rounded and reacted neither to light nor to accommodation. The eyegrounds were negative. His speech was dysarthric with occasional approach to explosiveness and his voice was distinctly high pitched. The tongue was not deviated when first protruded though it was afterward constantly shifted from side to side. The soft palate and pharynx appeared functionally normal. During the entire consultation the patient emitted plaintive grunting sounds, which manifestations may, however, have been purely hysterical features as the man had always been a rather high strung nervous individual. This symptom was not present at subsequent examinations. There were no disturbances of superficial and deep sensibility nor of the special senses and the general physical findings were negative. The intellectual faculties were absolutely intact.

Course.—I did not have the opportunity of seeing this patient again until Sept. 23, 1919. He then complained of transitory pain and soreness on the top of the head and was still restless, uneasy and mentally distressed by his prolonged illness. Physical examination revealed, however, a striking amelioration. His motor strength had largely returned, his grasp now registered 120 and 110 with the dynamometer, his facial palsy had entirely cleared up and his gait was normal. The pupils, however, remained totally reactionless, speech was still defective with a tendency to explosive features and he continued to exhibit the slow unceasing contortions of the face and the restless movements of the left leg and foot. The grimaces and head displacements had all the characters of genuine athetosis whereas the movements in the extremities were more closely akin to choreiform disturbances.

He was seen again Nov. 12, 1919, Jan. 12, and April 16, 1920, and his condition had not materially changed except as regards steady improvement in general strength and nutrition.

Case 2.—L. S., a man 27 years of age, married, employed in a cigar factory, born in Poland, which he left when still young, could give no definite information concerning his family history.

History.—Aside from the ordinary diseases of childhood, he had always enjoyed excellent health, was a diligent worker, used tobacco in moderation and denied alcoholic indulgence and venereal infection. He was sent to me, Oct. 10, 1919, by a former pupil for an opinion regarding the nature and prognosis of very severe muscular twitchings which had developed progressively after an obscure meningeal disorder associated with severe mental derangement. The history of the events which led up to his present condition was supplied by his physician who stated that the patient had had a severe attack of influenza during November, 1918, from which he recovered with extreme slowness. Early in January, 1919, he rapidly developed acute mental disturbances with maniacal agitation and during three weeks was absolutely out of his mind and had to be constantly watched and frequently restrained. A detailed account of all the features of this illness unfortunately could not be obtained except the fact that this transitory mental disturbance was accompanied throughout by fever, rigidity of the neck and frequent jerky movements in the head and extremities. Meningitis was suspected and lumbar puncture performed. The fluid was clear, contained many cells and gave a strongly positive globulin reaction but contained no organisms. The Wassermann reaction was negative. He improved after the lumbar puncture and his psychic manifestations rapidly subsided but in the latter part of February, about three weeks after the cessation of his febrile and delirious state, he began to develop almost constant choreiform movements which became practically generalized.

Examination.—Neurologic examination, Oct. 1, 1919, disclosed as the most striking and prominent feature decided spasmodic excursions of the patient's head consisting of constant, rather coarse and ample displacements, somewhat resembling those of Huntington's chorea though far less rapid in execution. The head was rotated from side to side and especially obliquely hyperflexed on the chest then obliquely hyperextended backward in such a way that during the extensor phase the ear was approximated to the shoulder and the face turned upward toward the opposite side, thus recalling the appearance seen in spasmodic torticollis. At the same time the face exhibited the most diverse contortions, such as slow winking and frowning movements, retraction of the corners of the mouth or puckering of the lips, occasionally interrupted by more rapid myoclonic twitchings of the entire musculature of the face, particularly on the right side. The right arm and leg likewise exhibited almost constant movements, but these were relatively less striking, were almost confined to the distal segments of the extremities and consisted of pronation and supination of the forearm and hand, flexion and extension at the wrist, flexion and extension as well as abduction of the fingers, adduction and elevation of the foot and flexion and extension of the toes. There were no gross movements at the larger joints of the extremities but occasional shrugging of the shoulders and irregular oscillations of the trunk were visible when the patient changed from the recumbent to the sitting posture or from the latter to the standing position.

There was no Romberg sign and the gait presented nothing unusual though the patient maintained that he frequently could not control his steps, felt inclined to pitch forward and sometimes walked like a drunken man. As usually occurs in ordinary chorea the involuntary movements in the extremities stopped completely or almost when the patient executed commands, and members of his family stated that all twitchings usually ceased during sleep. It was difficult to designate correctly the spasmodic movements observed in this patient; they had nothing in common with the rhythmic myoclonic manifestations seen in various parts of the body in a fair percentage of cases of epidemic encephalitis; they borrowed the features of both chorea and athetosis and for this reason it has seemed more practical to label this form the choreo-athetoid type. In reality, however, confusion existed mainly as regards the twitchings in the extremities which greatly resembled choreiform movements though they were much less rapid and jerky than the latter and recalled by their relative slowness and labored execution the usual appearance of athetoid spasms. The spasmodic phenomena exhibited by the head and face were, on the contrary, absolutely identical with those seen in typical cases of bilateral and hemi-athetosis.

There was no motor paralysis in the extremities, but a certain element of rigidity was demonstrable more especially on the right side. With Mathieu's dynamometer the grasp was 120 on the right and 100 on the left. The tendon reflexes were very active but of symmetric intensity and were not accompanied by clonus nor by Babinski and Oppenheim phenomena. The tongue was protruded in a jerky manner and rolled from side to side or curled up in the roof of the mouth. The soft palate and pharynx appeared normal and phonation was perfect but speech was somewhat defective with frequent approach to stuttering or explosive in character, perhaps by reason of the unceasing labial spasm. A definite palsy of the right lower facial nerve was evident. Ocular excursion, pupillary responses and eyegrounds were normal. The special senses, general sensibility and sphincter control were undisturbed. The patient was anxious regarding the outcome of his affection and distressed by his enforced inoccupation but presented no evidence of mental deterioration. His mode of behavior, attention, memory, and judgment were all carefully studied and there appeared no residue of his previous psychic derangement. The heart, blood pressure, blood and urine examinations yielded negative findings. The patient had come from a distant city and unfortunately was never seen again despite repeated endeavors to locate him.

Case 3.—F. R., a married housewife, 34 years of age, had had no children and no miscarriages, and aside from a rather prolonged attack of chorea at the age of 14 and recurrent tonsillitis her past history presented nothing of unusual interest.

History.—During the latter part of February, 1919, she had a moderately severe influenzal infection from which it was claimed she never fully recovered. Early in June, 1919, she began to have pains in the back, neck and shoulders, felt weak and unsteady on her feet, complained of not seeing objects clearly, seeing double whenever she directed her gaze to the right and having distaste for all food. Within a few days she developed slight fever, muscular twitchings in the neck, hands and left hypochondrium and became so drowsy that her family physician became alarmed and requested a consultation.

Examination.—When first seen by me, June 10, 1919, the patient lay perfectly quiet in bed apparently in a peaceful slumber with flushed face, moist skin, rather slow and irregular respiration and a temperature of 101.2 F. She could be aroused without difficulty, executed commands quite well and answered all questions intelligently. She stated that she no longer had pains anywhere except in the back of the head but felt terribly tired and saw everything through a heavy veil or mist. She remained perfectly immobile during questioning except for occasional rather rhythmic and shocklike twitchings in various parts of the body, notably in the neck, left arm and abdominal wall. The cervical spasm tilted the head backward and to the left; in the arm the main movement occurred at the wrist and consisted of spasmodic flexion of the hand resembling cortical spasm, while in the abdomen there was rhythmic recession of the left hypochondrium somewhat analogous to what takes place in hiccough. Rarely, similar though less typically myoclonic twitchings were likewise seen in the left leg or on the right side of the body. These various spasms were rapid and jerky like those of chorea but differed from choreiform manifestations by reason of their relative uniformity or rhythmic character. There was a faint drooping of the eyelids and a distinct though asymmetric bilateral prosopoplegia, the right facial musculature being more involved than the left. The physiognomy presented a fairly typical Parkinsonian mask perhaps conditioned by the bilateral facial weakness. The eyes followed the finger in all directions but excursion was slow and attended by definite nystagmus, both horizontal and vertical. The pupils reacted sluggishly and imperfectly to light and to convergence. The eyegrounds were distinctly congested and the disk outlines hazy. There was slight dysarthria but no involvement of the tongue, soft palate or pharynx could be detected. The extremities were free from actual paralysis but a tendency to generalized rigidity was present and the tendon reflexes throughout were hyperactive and equal on the two sides. There was neither Babinski nor Oppenheim phenomenon. The arms presented no evidence of either ataxia or adiadokocinesis but an intention tremor appeared when the patient carried the finger tip to the nose or attempted to take a small object from the examiner's hand. General sensibility, special senses and organic functions were undisturbed. Owing to the gravity of the patient's illness it seemed unwise to get her out of bed for the purpose of testing her gait, but it was learned from the family that for two or three days prior to taking to her bed the patient walked in a very irregular fashion, her body swaying from one side to the other. Lumbar puncture was advised but refused. Three days later the patient's condition was decidedly worse, the drowsiness greater, the fever higher, the twitchings more accentuated and constant and furthermore nocturnal agitation with hallucinatory manifestations appeared. It was then decided to proceed with lumbar puncture. The fluid came out under considerable tension, was perfectly clear, contained 56 cells to the cubic millimeter, gave a strongly positive globulin reaction, a negative Wassermann reaction and an indefinite gold curve. The blood showed a leukocytosis of 17,000 and yielded a negative Wassermann reaction. The patient's condition seemed to have been materially improved in every respect, but the involuntary spasmodic phenomena persisted. A second lumbar puncture, performed June 20, 1919, showed a cell count of 35 and a still markedly positive globulin reaction.

Course.—From this time on steady amelioration occurred and the further history was supplied by the physician in charge who stated that within a week the temperature became normal and remained so. The drowsiness, ptosis, nystagmus and dysarthria disappeared within three weeks and by that time the patient had been allowed to be up and about the house. She continued, however, to present her peculiar jerkings of the head and the restless movements of the hand which had rather increased. Her convalescence was completed in the country and I unfortunately did not see the patient again until several months later.

The patient finally reported at the office, Dec. 15, 1919, in a state of profound mental depression because of the uncontrollable twitchings which practically incapacitated her and rendered life intolerable. To my great surprise her spasmodic manifestations had not only very decidedly increased in severity but had moreover assumed quite a different character. The musculature of the neck and shoulders was the seat of a most extraordinary and persistent spasm. The patient's head was rigidly hyperextended, then slowly rotated from side to side and at the same time deeply buried between the spasmodically elevated shoulders, giving the impression of an attempt at telescoping the head through the trunk. The spasm, which was essentially tonic in character, lasted two or three minutes, evanesced, then reappeared after an interval of from a few seconds to a minute and was accompanied during the greater part of its exhibition by tonic bilateral contraction of the platysma myoides, more marked on the right side. Had I not seen this patient during the acute phase of her malady, I would have been much inclined to look on this cervical spasm as a purely hysterical phenomenon. However, the other twitchings also had persisted and were plainly in evidence. During the spasmodic excursions of the head, which was more commonly lowered toward the left shoulder with consequent deviation of the chin toward the right, the facial musculature likewise participated in the elaboration of the twitching phenomenon, being animated by irregular pouting of the lips and frowning movements in the forehead. The left upper extremity, particularly the hand and fingers, constantly executed slow and irregular movements closely resembling athetoid spasm. The hand was pronated and deviated outward or hyperextended and the fingers alternately flexed and extended or spread apart, the thumb and index finger being apparently more involved than the other fingers. Only an occasional restless displacement was observed in the left foot. No contractions occurred at the larger joints of the extremities on the left side and none was seen anywhere on the right side of the body save the spasmodic shrugging of the shoulder previously noted. There was no evidence of motor weakness although an accurate evaluation of the grasp of the left hand was rendered impossible by the constant displacements of the fingers. The whole left side seemed distinctly more rigid than the right and exhibited more active tendon reflexes. There was neither ankle clonus, Babinski nor Oppenheim phenomenon. The patient was a highly nervous woman and developed a marked tremor throughout the upper extremities when asked to hold the arms quietly extended in front of her with fingers spread widely apart. A trace of ataxia was present in both arms. Ocular excursion was perfect in all directions and the pupils were rather small though yielding normal light and accommodation responses. The eyegrounds still showed rather full and tortuous vessels with otherwise negative disks. Examination of all of the other cranial nerves was negative save for the various muscle spasms previously mentioned. General sensibility, sphincteric control and trophic functions were intact. The heart dulness could not be satisfactorily mapped out owing to the patient's adiposity, but a loud systolic murmur was present over the apex and distinctly transmitted toward the scapula.

The patient was seen again Jan. 5, Feb. 26, and April 14, 1920, and while she thought that some improvement had taken place under administration of bromids and hyoscin hydrobromid, I could see no material change in her various spasmodic manifestations.

SUMMARY OF CHOREO-ATHETOID SYNDROME

All three cases included in the preceding series developed, within a period of three months after a typical attack of epidemic encephalitis, a syndrome characterized by choreo-athetoid spasms in the face, neck and extremities. The original manifestations of the encephalitis corresponded to the usual clinical type except in one case in which very severe mental disorder and maniacal agitation constituted the essential features of the symptomatology. None of the patients presented at any time evidence of external ophthalmoplegia, but in one case a permanent paralysis of the intrinsic ocular musculature was noted. In only one case was definite lethargy observed while in all three the facial musculature was distinctly involved either on one or on both sides. In a general way the so-called characteristic triad, lethargy, asthenia and oculomotor paralysis was reduced to its minimum expression in this series of cases. The predominating feature in all was the rapid and progressive elaboration of a choreo-athetoid syndrome essentially unilateral in distribution and remarkable for the exaggerated participation of the cervical and facial musculature. It is interesting that in all three cases more or less definite hyperkinetic phenomena of this same type were already recognizable in the initial phase of the disease. While the character of the twitchings at first was perhaps not absolutely pathognomonic, the spasmodic movements in the later or convalescent stage of the affection were fairly characteristic, being of the nature of athetosis in the face and neck and on the contrary more closely analogous to choreiform jerkings in the extremities. In Case 3, however, the spasms eventually assumed almost classic athetoid features, both in the neck and in the extremities.

CHOREOPSYCHOTIC TYPE

Case 4.—G. A., a married housewife, aged 38, was seen by me only once and her history was obtained from the attending physician on the occasion of a consultation held Oct. 17, 1919.

History.—Aside from what was regarded as a relatively mild and even doubtful attack of influenza in the early part of September, 1919, there was nothing of unusual importance in her history. The first symptoms of her illness had appeared from eight to ten days before I saw her and consisted mainly of severe pains in the left occipital region, in the back of the neck and left shoulder and radiating into the left arm. This was interpreted at first simply as neuralgia. Within two or three days, however, it was followed by mild fever, diplopia, peculiar nodding movements of the head, muscular twitchings in the left arm and rapidly increasing tendency to nocturnal agitation, mental confusion with marked disorientation and eventually by hallucinatory delirium. She was fairly quiet during the day although at no time lethargic and rarely slept more than an hour at a time. The physician and the two attending nurses stated that at night more especially the patient constantly tried to get out of bed, had to be closely watched, fretted about the care of her house and spoke of nothing except the necessity of personally directing the management of her household. She would sit up in bed, point her finger toward a corner of the room and, as though speaking to her servant whom she called by name, she would carry on an imaginary conversation, giving detailed orders for tasks which it was known she had previously planned to carry out. She would insist on definite answers and accounts, remonstrated, pleaded or approved, and continued in this manner a good part of the night. She was totally disoriented for time and place but recognized her doctor every time he came into the room and named him correctly. During the day she had lucid intervals when she seemed to realize that her mind had wandered the greater part of the time and she would herself call attention to the silly things she had said and done, but usually she would almost immediately lapse back into her dreamy and confused mental state. Her temperature had oscillated between 99.6 and 100, and never exceeded 101.6 F. She took nourishment rather reluctantly, had no difficulty in swallowing and retained control of the sphincters.

Examination.—When I saw the patient Oct. 17, 1919, she was lying on her back with half closed eyes and a dreamy countenance. The first fact which attracted my attention was the peculiar and practically constant nodding of the head. This consisted in a fairly rhythmic and rather jerky spasmodic rotation of the head to the right with very slight associated backward tilting. The left arm also exhibited constant irregular and jerky movements consisting of pronation and supination of the forearm and hand, flexion and extension of individual fingers with frequent picking at the bed clothes. Restless movements and displacements also occurred in other parts of the body but were much less frequent and noticeable. By talking rather sharply to the patient and constantly urging her it was possible to proceed with a fairly satisfactory neurologic examination. At rest there was slight drooping of both eyelids and an appearance of mild divergence of the eyeballs but the patient could be made to follow the finger in all directions. No definite ophthalmoplegia was evident but a well marked vertical nystagmus was present and the patient claimed that she saw double whatever the direction of her gaze. The pupils were small and reacted sluggishly to light. The eyegrounds were distinctly congested and the outline of the right disk poorly defined. The facial musculature was flaccid on both sides and showed appreciable paresis. There was no gross paralysis in the extremities and all movements were executed but the left arm appeared diffusely paretic and atonic. Both arms presented slight ataxia and well marked intention tremor. When the patient sat up in bed the nodding of the head became less constant and was largely replaced by a coarse intention tremor such as occurs in advanced cases of multiple sclerosis. The tendon reflexes were all hyperactive but without accompanying ankle clonus or Babinski-Oppenheim phenomena. The abdominal reflexes were abolished and the stroking intended to elicit them was followed by unusually pronounced dermographia. The tongue showed no deviation but was only imperfectly protruded and speech was slow, monotonous and definitely dysarthric though usually intelligible. The temperature at this time was 99.8 F., the pulse rate between 110 and 120 and the respirations rather shallow and irregular. Lumbar puncture yielded a perfectly clear fluid under moderately increased pressure. The cell count per cubic millimeter was 32, the globulin reaction strongly positive (+ + +), the Wassermann reaction negative in all dilutions and the colloidal gold curve negative.

Course.—Four days later I was informed by the attending physician that lumbar puncture produced no amelioration in the patient's condition. She continued her nocturnal agitation and hallucinatory manifestations but rapidly became more drowsy during the day, showed signs of increasing exhaustion, developed dysphagia and hypoglossal paralysis and died three days after I saw her from respiratory paralysis. Unfortunately a necropsy could not be secured.

Case 5.—J. W., aged 43, a man, married, a shipping clerk by occupation, was seen in consultation with his physician, Jan. 30, 1920.

History.—Aside from an attack of sunstroke several years before, his past history was exceptionally good and he had always been a perfectly healthy and active man. No influenzal infection preceded the present illness which began, January 24, with paroxysms of pain in the occipital region and diplopia. The latter symptom annoyed him so much that he had all the pictures on the wall of his room removed. His temperature rose to 101 F., and remained at that point for a few days; the diplopia disappeared after three days, but the occipital pains persisted and at times gave him considerable distress. There was no paralysis anywhere at this time according to his physician and the superficial and deep reflexes were undisturbed. The patient soon assumed a drowsy and passive attitude and would lie for hours with eyes closed, totally indifferent to whatever took place about him and except for slight choreiform twitchings in the upper and lower extremities appeared perfectly quiet as though in a peaceful slumber. He could be aroused without difficulty, carried on conversation regarding the weather conditions, his state of health, etc., but soon drifted into details pertaining to the work in which he was engaged prior to the onset of his illness. His work seemed uppermost in his mind and he frequently muttered something in regard to it, became markedly disoriented for both time and place, but recognized those about him and called them by name though he took no further interest in their doings and never passed any remarks when letters received from a member of his family who had just left for a neighboring city were read in front of him. At night especially, though sometimes during the day, he became more markedly confused and restless, would sit up in bed and talk incoherently for long periods, almost invariably referring to some phase or other of his occupation and exhibiting by both word and gesture unmistakable evidence of hallucinatory delirium. The necessity of carrying out some given task seemed all absorbing and he frequently insisted on getting hold of a box he had to fix. He tried to get out of bed on several occasions but was never violent and could always be induced to lie quietly and obey the doctor's orders.

Examination. — When I first examined this man, January 30, I was surprised to find relatively little objective evidence of serious organic disease. He was rather somnolent but easily aroused, answered questions quite well, executed all commands and appeared perfectly rational. He readily lapsed back, however, into his habitual state of indifference and mental inertia. Restless and jerky movements were observed in the extremities but only at intervals and consisted in the arm of flexion at the elbow, flexion at the wrist and ulnar deviation of the extended and abducted fingers. In the lower extremity abduction of the thigh and extension of the foot and toes predominated though at times the whole limb was the seat of a more violent kicking movement. These various twitchings had a jerky spasmodic character and involved all four extremities but were rather more frequent and pronounced on the right side. No analogous twitchings were seen in either the face or neck. A very faint hemiparesis was present on the right side affecting the lower facial region as well as the extremities. Otherwise no paralytic disturbances were observed. All of the cranial nerves were intact and not the slightest implication of the ocular muscles could be detected. The pupils were small, in fact almost myotic, but nevertheless yielded normal light and accommodation reflexes. The cutaneous reflexes were rather sluggish but the tendon jerks were readily elicited though without appreciable exaggeration. The plantar reflexes were normal. No disorders of general or special sensibility were discovered and sphincter control was perfect except during a period of twenty-four hours at the beginning of the second week of the illness when the patient had to be catheterized. Examination of the eyegrounds disclosed negative findings.

Course.—This was the mildest case of the series and the entire symptomatology consisted at this time of more or less drowsiness, fairly persistent mental confusion, disorientation with episodic hallucinosis and jerky twitchings in the extremities. Lumbar puncture gave a perfectly clear spinal fluid with a direct cell count of only 15 per cubic millimeter but a strongly positive globulin reaction (+ + +). The Wassermann and colloidal gold reactions were negative. Unfortunately no blood examination was made. The urine showed no abnormal constituents. The patient was apparently markedly benefited by lumbar puncture and was much clearer mentally and quieter physically for three or four days, after which the former state of affairs gradually reappeared. He was seen again, February 13, and seemed to me much less drowsy than on the previous occasion and less restless and jerky. A second lumbar puncture was performed, the cell count was reduced to 7 and the globulin reaction was only faintly positive (+). From this time on the patient showed rather steady improvement in all respects and during the fifth week began to sleep in more natural fashion and at more conventional hours. The pseudochoreiform twitchings gradually subsided and he took a more active interest in his family and surroundings. Nothing further of importance was recorded by his physician. The patient at my request reported at the office for examination, April 22, and a complete survey of the neurologic functions was carefully made. He complained of paroxysmal twinges of pain in the right suboccipital area and right side of the face but apart from infrequent winking of the eyes and very defective wrinkling of the forehead and elevation of the upper lip, all of which gave him a rather definite Parkinsonian facial appearance, absolutely no objective evidence of residual disturbance was detected. No twitchings were observed anywhere and his psychic faculties were intact. He was considered to have virtually recovered.

Case 6.—T. G., a man, 45 years of age, married, assistant manager of a contracting and building concern, an Italian by birth, a rather high strung and impulsive individual, was considered an unusually able business man, used tobacco rather excessively but aside from light wine at occasional dinners never indulged in alcoholic stimulants.

History.—With the exception of the ordinary diseases of childhood and typhoid fever at 20, he had always enjoyed remarkably good health and was steadily active at his work. In the early part of December, 1919, he contracted a severe cold and subsequently developed influenzal manifestations with fairly sustained high temperature and apparently bilateral bronchopneumonia. He was confined to his bed for about three weeks and took fully another month to regain his strength and energy. He came home, Feb. 20, 1920, complaining of lassitude, headache, dizziness, pains in the back and legs and blurred vision. His family physician was called, found no fever but nevertheless ordered him to bed and gave him a calomel and saline purge together with aspirin. The following evening he felt much better and planned to return to his office on the succeeding morning but not being permitted to do so became greatly exercised and agitated, abused the doctor, refused to eat and quarreled with every one in the house. From this time on he rapidly developed quite a striking series of motor and psychic disturbances, became restless, paced the floor a large part of the time, showed tremor in the hands and irregular facial twitchings, would not go to bed and constantly talked of gigantic building plans which would place him at the head of the largest building corporation in the country. During the night he would occasionally lie down for an hour or so but even then continually muttered to himself or called out in a loud voice, giving orders to his subordinates and carrying on lengthy conversations with his associates. At no time did he show any evidence of drowsiness or apathy.

When I saw the patient in consultation, March 2, 1920, he was still restless but perfectly manageable and even morbidly good-humored though he became antagonistic the moment he learned the purpose of our visit. He persisted in saying that he did not need the services of a physician, that he was not ill, had never felt better, in fact, but that nobody understood him, that his own people were against him, believing him mentally unsound when in reality he was conceiving great schemes which were bound to triple the business of his firm and lead to his own advancement. He confided to the doctor that he had been most fortunate in recent speculations, had large sums stored away in some New York banks and that he contemplated building a magnificent residence in the suburbs of the great metropolis where his wife and two daughters would finally have the opportunity of entering the world of wealth and culture to which they belonged. This and similar grandiose ideas were the dominating features of his mental operations and evidently accounted for his visible exuberance and the outbursts of laughter which frequently interrupted our subsequent examination. During most of this introductory conversation he either stood up, shuffling his feet, or paced the floor; occasionally he sat down, but some part of the body was constantly in motion and one was instinctively reminded of what Charcot called "choreiform instability." One foot or the other was pushed forward or retracted, the heel or the forepart of the foot was displaced mesially or laterally, the knee was repeatedly adducted and abducted, the hand and forearm were slightly pronated and supinated, the hand was extended and irregular drumming or grasping movements of the fingers were executed. These various movements had a jerky and spasmodic character, and occurred on both sides of the body but were more pronounced on the right side. The head was frequently turned to one side, but without spasmodic element, and facial twitchings consisting especially of a clonic quiver of the cheeks and elevation of the eyebrows were almost constant. The facial contractions were much accentuated when he was requested to follow the finger with the eyes, close the eyes or elevate the upper lip. The twitchings in the extremities on the contrary diminished when the patient carried out designated movements and were replaced by a coarse tremor.

Examination.—He was a rather thin but muscularly developed individual of medium stature. The various tests for motor functions revealed no paralysis in the extremities but a marked and rather coarse intention tremor appeared whenever he held the extended arms directly in front of him or carried the finger to the tip of the nose. A similar tremor was present in either lower extremity on elevating it to touch the examiner's hand with the big toe. The tremor did not progressively augment in amplitude as is usual in multiple sclerosis, and no associated ataxia or dysmetria was observed. His grasp was powerful and equal on the two sides. The tendon jerks were all hyperactive but unaccompanied by either clonus or pathologic responses of the great toe. The umbilical and cremasteric reflexes were retained. The cranial nerves presented only slight abnormalities. There was a definite ptosis on the left side and the facial musculature on that side was relaxed, the angle of the mouth drooping distinctly on elevation of the lip. Ocular excursion was excellent in all directions save for convergence which was practically nil, and a faint nystagmus appeared when the eyes were carried to either the extreme right or left. The pupils were small, perfectly rounded, equal in size and reacted neither to light nor to accommodation efforts. Examination of the eyegrounds was negative. The tongue was protruded well and without deviation, but showed a fine fibrillary tremor at the tip and along the lateral borders. The closed eyelids presented a marked tremor. Speech was not materially affected, but occasionally the patient seemed unable to find his word, stuttered and mispronounced the word particularly if asked to repeat a difficult one. There was no involvement of the organic sphincters and no disorders of general or special sensibility.

In many ways the physical examination was unsatisfactory and many tests could not be properly carried out owing to the patient's psychic condition. His attention was easily diverted and it was plain that his mind was elsewhere, deeply engaged in the pursuit of his delusional consolations. Moroever, he constantly interjected sarcastic remarks regarding the examination, cracked jokes about doctors in general, or indulged in loud and prolonged laughter. This was clearly akin to what has been described as "witzelsucht" or "moria." His memory was shown by careful control to be grossly defective and he gave a most erroneous account of certain important incidents which had interested him prior to his influenzal infection.

Evidently the clinical physiognomy of this case was so strongly suggestive of general paresis that diagnosis was impossible without further observation and laboratory tests. Lumbar puncture yielded a perfectly clear fluid under moderate tension giving a cell count of 34 per cubic millimeter and a well marked butyric acid reaction for globulin (++). The colloidal gold curve in this case showed a definite discoloration in the first four or five tubes, but the Wassermann reaction was negative in all dilutions. The blood Wassermann reaction was likewise negative. The leukocyte count was 11,000 and the urine practically normal in every respect.

Course.—The patient was rather more quiet, both physically and mentally, during the two days following lumbar puncture, but soon he began to exhibit the same motor unrest, irregular jerky movements and delusional manifestations of grandiose type. His various symptoms did not materially change until approximately two weeks later, when he suddenly became semicomatose after a night of unusual agitation with prolonged shouting and wild delirium. When seen on this occasion, March 18, he was profoundly somnolent, could barely be aroused, merely muttered unintelligibly in answer to questions, would not protrude the tongue nor squeeze the hand, but nevertheless swallowed food placed in his mouth. His breathing was rapid and irregular, occasionally slower and stertorous, his pulse was rapid, oscillating between 120 and 130, but perfectly regular, and his temperature by axilla 100.2 F. This time he again almost constantly presented rapid and irregular jerky displacements of his extremities and facial twitchings. This stuporous state lasted a little more than a day and was followed by a recrudescence of the hallucinatory delirium and delusional wanderings. He was totally disoriented, no longer seemed to recognize those about him, refused medicine and sometimes food, talked incoherently the greater part of the time and lost all concept of propriety, voiding in bed or on the floor. A similar stuporous attack appeared March 24 and lasted almost three days. Lumbar puncture performed at this time again gave a perfectly clear fluid with a cell count of 60 and a strongly positive globulin reaction. Although considerable fluid was withdrawn, the procedure did not modify the patient's condition in the least and when he regained consciousness he at once started to display his previous motor agitation, mutterings and absurd ideas. He was, moreover, perceptibly weaker, seemed to use the right extremities in a weak and ataxic fashion and could be more easily restrained. He took little nourishment, swallowed with some difficulty and in a few days gradually became comatose for the third time, developed Cheyne-Stokes' breathing, paroxysmal hiccough, marked dysphagia and a rising temperature. The spasmodic jerkings eventually subsided and he died, April 4, after three days of coma.

Necropsy.—A limited necropsy was obtained, examination of the brain only being permitted, and we had to be content with removal of fragments for microscopic examination. However, enough information was secured to establish positively the anatomic diagnosis.

The brain was remarkably edematous both on the surface and on section and the leptomeningeal vessels at the base and over the frontoparietal region were distinctly engorged. The histologic findings corresponded to the lesions thus far described by other observers. There was only discrete infiltration of the pia with mononuclear cells, whereas the intracerebral vessels, more particularly the veins, were markedly distended and showed extensive adventitial infiltration. A diffuse interstitial infiltration was likewise present, but no definite glia hyperplasia was detected. Minute hemorrhages were found in the midbrain and corpus striatum, but were particularly abundant in the subcortex of the left frontal lobe. There were no advanced degenerative changes in the ganglion cells, but merely rarefaction with imprecision of the tigroid substance and a more or less diffuse staining reaction. These various lesions were practically identical at all levels examined, but seemed more severe and extensive in the cortex and particularly in the subcortex of the frontal regions. It was in this area that the vascular changes were most striking and the pial infiltration most pronounced. The larger pyramidal cells were largely deprived of chromatin granules and many were surrounded by the infiltrating cells. Nowhere did the blood vessels exhibit the characteristic structural modifications found in syphilis and paresis.

Case 7.—I. W., a man, 40 years of age, married, a tailor by occupation, was admitted to the public wards of the Albany City Hospital, March 29, 1920, complaining of nervousness, weakness and pains in the neck, back and in both arms from the elbows to the tips of the fingers. These pains had occurred in paroxysms since a severe cold contracted March 8, 1920.

History.—His family and past history were unimportant. He stated that he had enjoyed relatively good health, had always been a hard worker, slept little, used alcohol in moderation and had never contracted any venereal infection.

The present illness began about March 8, 1920, when he contracted a severe cold lasting several days, during which he felt feverish and fagged out. Then paroxysmal intense pains appeared in the neck, back, chest and upper extremities. At the time of admission he stated that the pains were no longer of great severity and that numbness and tingling in the arms and hands were the most annoying symptoms. General physical examination by the house physician revealed nothing of serious nature and his temperature was normal, but he appeared nervous and restless and exhibited occasional facial twitchings and jerky movements in the extremities. Two days later he began to show evidence of mental confusion and disorientation, especially at night, and developed involuntary and incoordinate muscular twitchings throughout the body, but more particularly in the facial and abdominal musculature. His skin was unduly moist, his face pallid and his pulse rapid, ranging from 100 to 130. His symptoms rapidly became more accentuated and mental confusion, marked disorientation for time and place, together with motor unrest, appeared, especially at night, alternating by day with periods of drowsiness during which he muttered incoherently. At night it was impossible to keep him in bed; he wandered about the ward, got in bed with other patients, crept about the floor engaged apparently in finding some lost object, entered the lavatory and laid out towels and cloths as though in the act of folding or pressing clothes. When asked what he was doing he retorted that he was a tailor and had to do his work. The blood showed a leukocyte count of 12,000. The urine showed a trace of albumin and a few finely and coarsely granular casts.

Examination.—At the neurologic examination, April 1, 1920, the patient was found lying relatively quiet in bed with no indication of the nocturnal tendency to irrepressible agitation. He did present, however, frequent facial twitchings and contortions and almost constant and definitely choreiform movements in the hands and fingers as well as in the lower extremities. There were no gross displacements of the trunk nor coarse and ample movements at the larger joints of the extremities, but one finger after another was flexed, extended or abducted, the hand flexed, extended or supinated, the knee adducted or abducted, the foot flexed and adducted or extended and the toes similarly displaced. Otherwise he looked drowsy and apathetic and his facial expression was distinctly masklike. The eyes followed the finger in all directions and no ophthalmoplegia could be detected, although at rest a slight divergent strabismus was evident, perhaps more marked on the right side. The pupils were small and reacted sluggishly to light and convergence efforts. The imperfect elevation of the upper lip suggested a faint paresis of the facial nerves. The tongue protruded well and without initial deviation, although it was constantly moved about, presumably participating in the general choreiform instability. There was no motor paralysis in the trunk and extremities, but the grasp was decidedly weakened, more so on the right side. The tendon reflexes were all present but hypo-active. Neither the Babinski nor Oppenheim phenomenon was present. The cremasteric and abdominal reflexes were not obtained. The sphincters were perfectly controlled. When the patient assumed the sitting or erect posture he presented a distinct intention tremor of the head. The gait was not characteristic although the patient occasionally took uneven steps and on two or three occasions showed a slight titubation. His speech was dysarthric, but by reason of his foreign birth and limited education it was difficult to interpret correctly this particular detail. At times rapid utterances were followed by slow, hesitating, almost stuttering enunciation. He exhibited amnesia for both remote and recent events, but especially for the latter, was not clearly oriented but never appeared antagonistic in the day time and obeyed all requests during the course of examination. The blood Wassermann reaction was negative. The spinal fluid was clear, gave a cell count of 25 per cubic millimeter and a strongly positive globulin reaction by Pandy's method; the Wassermann reaction was negative with 0.5 c.c. The colloidal gold reaction gave the interesting curve 5555421000.

Course.—April 3, 1920, the patient was transferred to the psychopathic pavilion as he no longer could be managed in the general ward. From this time on he exhibited progressively more striking changes in the mental sphere; he continued to wander about at night and to get in bed with other patients. One dominant idea seemed persistently to absorb his mind and command his activities and this was the necessity of pursuing his usual work as a tailor. He repeatedly stated that he was working at his trade and this imperative concept probably explains the fact that he constantly was found busily engaged in definite tasks with his bed clothes which he either spread out carefully on the floor, wound about his person or tied up in bundles. At other times, however, his aberrations migrated into other channels and he ran about the rooms talking incoherently or laughing and singing as though in jovial mood. Night after night he crept out of bed and could not be induced to return to it, although only once did he develop any tendency to wild delirium and opposed strong resistance to physical restraint. On one occasion he was found sitting beside his bed muttering to himself in a low tone and when asked why he did not go to bed he stated that there was no place for him, the bed was filled with babies, he could see, feel and hear them, he knew they were not his own children, but of course he would not disturb nor harm them. This state of affairs continued without much change during two weeks, but the patient was nevertheless losing ground physically as well as mentally despite the fact that he ate his meals without urging and even seemed to relish his food. During this period his temperature oscillated between 99 and 101 F., he slept less and less even in the day time, showed increasing motor weakness, looked haggard and utterly exhausted and betrayed evidence of mental depression. His muscular twitchings and choreiform movements rather decreased in severity and his speech became almost unintelligible. Otherwise the neurologic findings were not materially altered. Lumbar puncture done April 6 gave again a perfectly clear spinal fluid which showed a cell count of 23 per cubic millimeter, a well marked globulin reaction by the Pandy and Noguchi methods and a negative Wassermann reaction with 1 c.c. The colloidal gold curve this time was faintly marked and not characteristic. Another examination of the spinal fluid, April 12, showed the cell count to be 150 (?) per cubic millimeter and the globulin reaction still strongly positive.

During the week of April 18 an erythema appeared on the lateral aspect of the right arm and rapidly became infected owing to persistent scratching by the patient so that evidence of a complicating lymphangitis soon made it imperative to keep hot packs over the arm. As the patient constantly pulled off the dressings it became necessarry to keep him in bed and restrain the arms. He rebelled against this measure, but his increasing motor weakness did not enable him to oppose much resistance. The jerky restless movements which had previously diminished in severity again appeared in much accentuated form and could not be interpreted as efforts to get out of bed as there were no coarse truncal displacements nor any attempts to place the unbound lower extremities on the floor. The twitchings consisted of constantly varying movements of the hands and fingers as well as of the feet and toes, together with adduction and abduction of the knees; they were quick, jerky and irregular movements quite similar to those of chorea. The patient was sleepless, muttered a great deal, but his speech had become absolutely unintelligible. April 24, his physical condition remained practically unchanged although he exhibited a new mental attitude, seemed morbidly jovial, amused by our questions and tests and repeatedly laughed aloud for several seconds as though in derision of the situation. He obeyed simple orders such as showing the tongue or squeezing the hand, but could not be induced to follow the finger with the eyes nor to relax the limbs for estimation of the reflexes. The whole body was rigid and cold, the temperature subnormal, the pulse rapid, ranging between 110 and 130, the tongue heavily coated and the sphincters apparently incontinent. The pupils were moderately wide, but responded actively to light, the tendon reflexes were not elicited, owing presumably to the rigidity. Despite this alarming state of affairs the patient continued to take nourishment satisfactorily and held his ground without much further change until May 5, when he began to show distinct evidence of substantial amelioration. He became more quiet, exhibited less confusion and disorientation, articulated more distinctly and the jerky twitchings of the extremities greatly subsided. A complete neurologic examination practiced May 8 showed a surprisingly excellent condition. The patient answered questions readily, executed commands promptly, appeared perfectly oriented as regards time, place and person, correctly estimated the length of his stay in the hospital, conversed intelligently about his family, his occupation and the onset of his illness and asked how long it would be before he would be allowed to go home. He still exhibited slight jerky twitchings in the hands, knees and feet, but was no longer rigid and could execute all the ordinary movements with the arms and legs. The whole right side seemed slightly weaker than the left and the tendon reflexes on that side were appreciably more active. There was neither Babinski nor Oppenheim phenomenon. The facial musculature on both sides was paretic, but ocular excursion was excellent in all directions, there was no ptosis and the pupils reacted actively to light and accommodation. Speech was still dysarthric but quite intelligible. Sphincteric control was perfect. His temperature had remained normal for ten days and he could now sleep relatively well.

SUMMARY OF CHOREOPSYCHOTIC SYNDROME

This group of cases, like the preceding series, was characterized by a minimum display of the ordinary and more familiar manifestations of epidemic encephalitis. Decided somnolence or relative lethargy was present only in Case 5 and in no case was well marked ophthalmoplegia observed although partial ptosis was noted in two (Cases 4 and 6). Some degree of facial weakness, however, existed in all. The most striking features in this series were the choreiform jerkings and the persistent psychic disturbances. While the twitchings cannot be said to have been absolutely identical with those seen in classic instances of Sydenham's and Huntington's chorea, they were more closely analogous to them than to any other form of hyperkinetic disorder. These spasmodic movements, for the most part at least, were neither coarse tremors, rhythmic myoclonia nor jactitations, but consisted of rapid, jerky, irregular and incoordinate displacements almost never occurring in more than one extremity at a time and for which the term "choreiform twitchings" appears to be the only appropriate designation. The psychotic manifestations included marked and persistent mental confusion, disorientation for time and place, hallucinatory delirium, more particularly nocturnal and delusional states apparently originating in part from the hallucinations. It is a notable fact that in every case the hallucinations and delusions were intimately related to the usual work or occupation of the patient, who seemed totally enslaved by the fixed idea of the necessity of pursuing his habitual activities. Case 6 is particularly interesting by reason of the remarkably accurate mimicry of the classic features of general paresis. Two of the four cases in this series terminated fatally, a point emphasized by others who have reported more or less analogous types of epidemic encephalitis.

CONSIDERATIONS ON PATHOGENESIS

The choreo-athetoid and choreopsychotic syndromes illustrated by the foregoing series of cases serve to substantiate the claim that in epidemic encephalitis the distribution of the lesions does not always limit itself to the mesencephalon and adjoining areas of the brain, and they likewise furnish an opportunity for tentative localizations in more minute or circumscribed fields within the massive area embraced by the striothalamotegmental region. While no positive conclusions can be formulated regarding the exact distribution of the lesions in the absence of satisfactory necropsies, an attempt at theoretic localization may not be unjustified on the ground that some of the symptoms observed have a well recognized anatomic basis and that much light has been thrown in recent years on the regional substratum of both choreo-athetoid syndromes and certain well defined mental disorders. In a valuable and instructive series of monographs, J. Ramsay Hunt[13] has recently contributed some very interesting personal observations on the pathology of the corpus striatum, particularly with reference to the pathogenesis of paralysis agitans, and at the same time he has collected and carefully analyzed most of the previously accumulated data bearing on such affections as athetosis, Huntington's chorea, progressive lenticular degeneration and allied syndromes. He concluded that there were two syndromes resulting from elective cellular degenerative changes in the corups striatum, namely chorea and paralysis agitans. While there is great fear that the lenticular nucleus, like the endocrine glands, is being unduly called on to assume the pathogenesis of all that remains obscure in the domain of neurologic interpretation, there can be no doubt that choreiform and athetoid disturbances are intimately related to minute lesions either within or bordering on the caudate and lenticular nuclei. Several years ago Marie and Lhermitte[14] reported several cases of Huntington's chorea in which they found chronic leptomeningitis, atrophy of the frontal lobes, dilatation of the lateral ventricles and massive atrophy of the corpora striata, of the putamen more particularly. These ganglions together with the frontal cortex presented well marked degenerative changes in the nerve cells, hyperplasia of the glia tissue and definite alterations of the vascular system. Marie and Lhermitte attributed the mental disorder to the meningocortical lesions, and the choreiform manifestations to the changes found in the corpora striata. Reasoning by analogy and having in mind what has already been established regarding the distribution of the lesions in epidemic encephalitis, it is possible to furnish a plausible explanation for the symptomatic groupings observed in the present series of cases. It is true, of course, that in epidemic encephalitis the lesions are too diffuse to warrant precise deductions, but if we assume, as seems perfectly legitimate, that the subacute or chronic course of some cases is due to slowly evolving changes, there is no reason why the meningeal congestion, adventitial lymphocytic infiltrations, interstitial edema, multiple hemorrhagic extravasations and cellular disorganizations occurring in this disease cannot give rise, according to the focal predominance of the lesions, in one case to a combination of pronounced psychotic disturbance and choreiform jerkings, and in the other case to the progressive development of well characterized athetosis or hemiathetosis. In the first instance we have in a certain measure an approach to chronic degenerative chorea differing from it of course by reason of the more abrupt development of the symptoms, the rather dissimilar physiognomy of the psychic component and the favorable or else rapidly fatal outcome. In the second case we have a clinical picture which resembles to a remarkable degree the classic examples of athetosis. It remains to be seen, however, whether such syndromes, choreoathetoid and choreopsychotic, apparently transitory or recoverable, do not recur, and whether those which fail to clear up are not eventually transformed into perfectly typical instances of the characteristic neurologic affections which they already so closely imitate.

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[13] Hunt, J. Ramsay: Progressive Atrophy of the Globus Pallidus (Primary Atrophy of the Pallidal System): A Contribution to the Functions of the Corpus Striatum, Brain **40**:58 (May) 1917; Tr. Am. Neurol. Assn., 1917; The Efferent Pallidal System of the Corpus Striatum: A Consideration of Its Functions and Symptomatology, Tr. Am. Neurol. Assn., 10, 1917; Primary Atrophy of the Pallidal System of the Corpus Striatum, Presented at a meeting of the Assn. Am. Phys., 1917; Clinical Types of Paralysis Referable to the Pallidal System of the Corpus Striatum, Tr. Assn. Am. Phys., 1918.

At any rate the association of slight hemiparesis, dysarthria and disorders of emotional control with the choreo-athetoid manifestations justifies the assumption that the lesions border on the internal capsule, the absence of objective sensory losses and hemianopsia largely warrants the elimination of serious thalamic involvement and in this way an intralenticular or paralenticular localization asserts itself as the probable underlying cause of the spasmodic choreiform and athetoid disturbances. Thus, the pathology of epidemic encephalitis serves to confirm in a certain measure the actually favored hypothesis regarding the striatal origin of choreic and athetoid spasms. Similarly, a predominating implication of the meninges and cortex of the frontal regions is believed to be responsible for the psychotic forms of epidemic encephalitis as well as for the psychotic components incorporated in the other clinical types of the disease, whether of the choreopsychotic or the usual midbrain type. This supposition is in keeping with the prevailing conception of the physiology and pathology of the cerebral cortex and finds additional confirmation in the strikingly paretic type of mental disturbance observed in Case 6. It has been known for a long time that in general paresis the maximum corticomeningeal changes are distributed over the frontal regions of the cerebral hemispheres.

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[14] Marie and Lhermitte: Les lésions de la chorée chronique progressive, Ann. de méd., No. 1, 18, 1914.

The occurrence of choreopsychotic and choreo-athetoid syndromes as clinical types or sequelae of epidemic encephalitis, coupled with the positive knowledge of chronic forms of this disease, opens up a wide field for retrospective meditation on the genesis of a number of diseases concerning which we still remain quite ignorant. One would think, in the first place, of the various known diseases which some of the clinical types of epidemic encephalitis more or less completely simulate, i. e., athetosis, paralysis agitans and even Huntington's chorea and Friedreich's ataxia. Despite the fact that both of the latter conditions are regarded as hereditary or familial diseases, absolutely identical clinical complexes are not uncommonly observed in which all evidence of familial character is wanting. Likewise to be mentioned in this connection are multiple sclerosis, hypothalamic and dysglandular syndromes and perhaps even dementia praecox and other psychoses. It is puerile, of course, to suppose that epidemic encephalitis is a new disease; only our knowledge of its epidemic occurrence is new and it is extremely probable that, like poliomyelitis, the disease has existed in sporadic form for centuries. This consideration may be helpful in our retrospective endeavor to supply a logical basis for the development of the various diseases named in the foregoing and which neurologists have been observing for years in more or less fully constituted form but without definitely ascertainable notions regarding the far remote initial disturbances.

A point of particular interest in connection with the symptomatology of epidemic encephalitis is the genesis of the variations in the clinical picture and consequently the mechanism underlying the regional variations in the focal predominance of the lesions. In an article on poliomyelitis,[15] I have previously expressed the opinion that the localization of organic cerebrospinal disease is not entirely accidental but on the contrary largely conditioned by predisposing functional exhaustion of certain areas or cell groups resulting either from prolonged physiologic hyperactivity or from antedating trauma or peripheral disease affecting the related innervation zones. This assumption implies that the natural defensive reactions at such levels are at low ebb and consequently that the fiber tracts or cell complexes concerned, offering inadequate resistance on the advent of a cerebrospinal infectious invasion, succumb first and most severely. The resulting clinical manifestation may be either paralytic or irritative according to the level at which the neuron is disabled and according to the character of the lesion as well as to the degree of involvement. This conception may not have the merit of originality and is doubtless shared by many others, but it is nevertheless a fact that such an interpretation has sufficient practical significance to warrant greater emphasis than it actually receives either in standard textbooks or in current literature. A case in point is supplied by the observation of a patient recently examined who presented a poliomyelitic type of epidemic encephalitis.

The case was that of a woman employed in a department store, who was constantly called on to lay on the counters merchandise stored away in a cellar. Several times a day she was obliged to lift or pull up a trap door in order to get the merchandise. March 20, 1920, she developed general pains, fever of mild degree, unusual somnolence and within a period of forty-eight hours there appeared a paralysis of the right upper extremity. When seen, April 2, it was found that the paralysis was not total, involved particularly the deltoid, spinati and pectoralis major and to a lesser extent the brachialis anticus, biceps, and supinators. The paralysis was atonic with much weakened tendon reflexes in the arm and poor responses to the faradic current. The right lower extremity was somewhat rigid and exhibited increased tendon jerks. This was clearly due to a lesion in the anterior horn of the cord at the level of the fifth and sixth cervical segments with infiltration of the outlying lateral column. When seen again, May 5, all that remained of these symptoms was a much weakened grasp and inability to abduct and elevate the arm much above the horizontal.

Cases possessing similar demonstrative value have been frequently observed. Bassoe[9] recently reported a case illustrating the "meningo-radicular" type of encephalitis in which a right sided facial paralysis of the peripheral type was noted in a patient who had had a facial paralysis on the same side three years before. In the present series of cases it is not particularly difficult to establish the etiologic relationship of occupational strain to the salient manifestations of the clinical picture. The traveling salesman (Case 1) who constantly hurried to make train connections, impatiently stepped back and forth watching out for late arrivals, indulged at all times in hasty conversation and strove invariably to present a cheerful and winning countenance to all, developed restless movements in the legs, dysarthria and facial contortions suggestive of efforts at distant accommodation. The cigarmaker (Case 2), who spent the major portion of each day seated with his fellow workers at a long table and who constantly turned his head to the right and to the left in conversing with his companions while busily engaged in trimming and rolling tobacco leaf, developed alternating flexor and extensor athetoid movements of the head and choreo-athetoid movements in the right forearm and hand. Regarding the patient figuring as Case 3, relevant muscular strain was apparently not concerned in the genesis of the spasmodic disorder, but, as was previously stated, the woman had had chorea several years before. The psychotic cases with one exception were observed in highly neurotic individuals of Jewish or Latin extraction who were known to have persistently worked either under considerable mental strain or overtime with altogether insufficient sleep and little opportunity for relaxation. That all of these patients continued during their illness the psychic labor associated with their usual activities was plainly revealed by the content of their hallucinations and delusions. With these several recent observations at hand and many analogous experiences in the past, one cannot help ascribing to this same factor of functional hyperactivity and focal exhaustion the preponderant involvement of the mesencephalon in epidemic encephalitis and the resulting frequency of lethargy and oculomotor paralysis. Probably never before have people slept as little and used their eyes as much as they have within the last five years. While the soldiers in the trenches were constantly on the lookout for bombs, shells and missiles of all kinds as well as for airplanes and advancing enemy detachments, the people at home hardly made a less heavy demand on the ocular musculature in persistently working overtime at the various tasks and industries directly related to the pursuit of the war. Never before have people in all walks of life so diligently read the morning and evening dailies, so frequently riveted their upturned eyes on war bulletins, so eagerly watched the evolutions of high soaring planes or so patiently stood before public buildings straining neck and eyes to get a glimpse of distinguished visitors and returning heroes. Let us now add to all this the very significant part played by motion picture theaters which are steadily cropping up everywhere in greater numbers and drawing ever increasing throngs. It is not that any one of these factors in itself has exerted a prejudicial influence but the combination of all of them operating over a protracted period has certainly implied fairly sustained hyperfunction of the motor nuclei of the midbrain and it is no exaggeration to state that at no time in the history of the human race have the accommodation mechanism and the associated movements of the eyeballs been brought more actively and persistently into play. It has been definitely shown that among the ocular symptoms of epidemic encephalitis by far the most common disturbances have been represented by ptosis, weakness of convergence with paralysis of accommodation and paresis of the associated movements of the eyeballs, more particularly of vertical excursion. Is it not infinitely more logical to interpret such distinctive ocular palsies as the outcome of the selective muscle strain emphasized in the foregoing than to attribute them to a supposed specific affinity of the encephalitis virus for the ganglionic complexes of the mesencephalon?

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[15] 15. Archambault: The Haematogenous Invasion of the Cerebro-Spinal Axis in Poliomyelitis, Alienist and Neurologist **39**:34 (Jan.) 1918.

CONCLUSIONS

1. Epidemic encephalitis is characterized by a remarkably protean symptomatology dependent on regional variations in the focal predominance of the underlying histologic changes.

2. Aside from the usual and familiar clinical picture of midbrain involvement, the disease may yield a number of clinical types closely resembling several well-known affections of the nervous system such as paralysis agitans, poliomyelitis, polyneuritis, cerebellar sclerosis, etc. The combination of choreiform twitchings and acute psychotic disturbance as the salient manifestations is not uncommon and choreo-athetoid or frankly athetoid syndromes may be observed as sequela of the disease.

3. The occurrence of a chronic or relapsing form of epidemic encephalitis may prove to have an important bearing on the origin of some of the affections which the clinical types of the disease simulate and of other ill understood conditions such as multiple sclerosis, dysglandular syndromes and certain psychoses.

4. Variations in the symptomatology of epidemic encephalitis are possibly due to individual variations in the relative susceptibility of different levels of the cerebrospinal axis and it is believed that regional exhaustion resulting from prolonged physiologic hyperactivity or from previous disease is an important factor in the localization of the lesions produced by infectious invasions of the central nervous system.

COMPULSION NEUROSIS IN A CHILD

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The following case of compulsion neurosis is related because of its occurrence in a child. At this period of life the most common of the psychoneuroses is anxiety, the primary cause of which is thought to be essentially physical. Where psychogenic causes are most operative in the formation of the neuroses, manifestations of these are rarely seen in childhood. The cerebration requisite for their formation is not ordinarily present at an early age; but when a neurosis does develop, the same psychogenic factors are as potent here as in the adult. This is well shown in the analysis by Freud of "Little Hans"[1] and in Jung's analysis of "Anna."[2] These cases are not compulsion neuroses, but their psychoneurotic symptoms have a basis similar to that present in the case reported.

REPORT OF CASE

Present Illness.—Jennie S. (No. 27390), aged 6 years, was brought to the Neurological Department of the Vanderbilt Clinic on Feb. 21, 1920, by her mother, who stated that she had noticed nothing abnormal in the child until Jan. 2, 1920. At this time she began to act peculiarly and said that she had pieces of glass in her mouth and in her hands. She cried and could not be reassured. Later she feared her hands were dirty and washed them every few minutes. She was afraid to touch any other child, and when her hand was touched she ran quickly to the sink to cleanse it. She also would not allow any one to touch her parents, and if they were touched she attempted to brush their clothes. On one occasion she would not touch a schoolmate, giving as the reason that she was afraid of her because the latter was near a hearse. She would not play with the other children of the neighborhood as she formerly had. One night she awoke crying and said to her father, "I think you are going to die." She seemed to be afraid to be with any one but her parents. The father, who accompanied the patient one week later, stated that on Feb. 20, 1920, she said (in Yiddish), "Papa, my bundle (vulva) is turning this way and that." She indicated the motion by pronation and supination of the right hand. Her father prohibited her from saying such "dirty things" and added, "It does not turn in a child," to which she answered, "Maybe yes and maybe not." One day later she came from school saying, "A boy touched my bundle." When castigated by her parents she said, "He nearly touched it." She perseverated such statements many times and later was also given to verbigeration; the exact words or phrases could not be obtained from the parents. The patient lately complained of pains in her head and arms, and also that her right leg "was turning." At times she complained of itching and was frequently observed scratching her back, legs and vulva.

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[1] Freud, S.: Analyse der Phobie eines 5jahrigen Knaben, Sammlung kleiner Schriften zur Neurosenlehre, Series 3, Leipzig and Wien., Franz Deuticke, 1913.

[2] Jung, C. J.: Ueber Konflikte der Kindlichen Seele, Jahrb. d. psychoan. u. psychopath. Forsch., **2**:33, 1910. Trans. by C. E. Long: Analytical Psychology, New York, Moffat Yard & Co., 1916, Chap. 2, Lect. 3.

Personal History.—The patient was born in Brooklyn. Delivery was instrumental after two days' labor; there were no injuries. At birth she had one tooth and two erupted soon after. She walked and talked at the age of 1 year. At 2 she had otitis media, and paracentesis of the drum was performed. At 4 years she had pertussis. Her habits as to appetite, thirst, bowels, urination and sleep were normal. She had no pavor nocturnus, nocturnal enuresis, thumb sucking or nail biting. Sexual proclivities were denied, with the exception of those mentioned above. She began school Jan. 2, 1920, in the kindergarten and seemed to make normal progress until the last few weeks.

Family and Social History.—The parents were Russian Hebrews, who had been in this country ten years; they were healthy and not neurotic. The father was 31 and the mother 26; they had been married seven years and patient was the only child. Mental and nervous diseases were denied in both ancestral branches. The parents' education was meager; they were of the working class. The father was a tailor and maintained a comfortable home. The patient shared the bedroom with her parents, and though she usually slept alone she was frequently taken into her parents' bed. She was very much pampered by her father, while the mother was the disciplinarian and appeared to be constantly on guard to correct her in brusque fashion even in the presence of strangers.

Mental Status.—On entering the examining room the patient hugged her mother closely, but she readily approached the examiner when he called her. She smiled good naturedly and though reticent at first, she was soon ready to engage in conversation. She cooperated well except that when she was undressed for the physical examination she showed resistance. Her mood was euphoric and playful. Her only mannerism was biting her upper lip continually. Her production was somewhat incoherent, at times irrelevant. For the most part she showed no hesitancy when speaking on neutral subjects, but there was marked blocking when her difficulties were broached. She stated that once while playing with glass lottos she took one into her mouth and grated her teeth on it. Later she believed that she had some chips of glass in her mouth and asked her parents to look for glass in her mouth. She cried because she feared she swallowed some glass. She denied ever having had any pains but, pointing to her forehead, she said, "At one time it turned." She talks as follows about her friends: "Eddie W. (does not give surname) chases all the girls. . . . I don't like him because he pushes the chairs all the time . . . the girls do it sometimes. When the teacher tells some boys to move the tables he says 'I'll move the tables.' Once a boy and girl were sitting and he runs like a horse and throws me down. He is so strong just like a horse." Here, in a whisper, she tells her mother that her teacher is married and asks if she can relate it. Her mother gruffly said "no," and though the examiner tried to prevail on the patient to talk, she refused to do so. About Eddie touching her she said, "I don't want to tell . . . because I am ashamed . . . I don't want to talk no dirty words." When asked why she talks to her parents about it she said "Because I am crazy . . . my mother told me so." She was oriented in all fields; no hallucinations or delusions could be elicited. In the Binet-Simon test she measured up to 7 years. For the association test stimulus words were selected from her production and neutral words were interspersed. To the neutral words her average reaction time was 2 seconds. The striking fact was the number of times she reacted with numerals, which was out of proportion to all her other reactions. Some of her associations are reproduced:

Stimulus Word	*Reaction*	*Time, Seconds*	*Reproduction*
die	I don't know	7	two
strong	to move tables . . . three	8.5	four cents
glass	tooth brush	6	three pieces of glass
Jennie	one	4	papa

Further associations were:

Three pieces of glass: Swallowed a piece of glass. . . . I don't know anything about it. . . . It is what we drink. . . . Glass is ten. . . . I count one, two three. . . I drink then eat breakfast then go to school. . . . After I get up from bed I make one and two. . . . One is better.

Strong: Eddie moves the tables. . . . He is strong. . . . He goes one, two, three (here she laughs and refuses to continue).

Physical Status.—She was a well developed and well nourished child of 6 years. No abnormalities were noted in the voluntary motor system. Gait, coordination tests and speech were normal. No tremor or choreiform movements were present. The reflexes present and normal. The pupils reacted to light and accommodation. There were no anomalies in the hair distribution and nails. There were no vasomotor disturbances except occasional cold hands. The heart and lungs were negative. There was an eczematous eruption on the hands. No serologic tests were performed. The glandular system was apparently normal.

Summary.—The patient, an only child, soon after she began school, developed queer actions which alarmed her parents She kept continually washing her hands, repeating words and phrases and expressing fears that she swallowed some glass, that she would burn and that her parents would die. She did not allow herself or her parents to be touched. She also complained of pains, headaches and turning movements of various regions of her body. Her conduct with her playmates changed; she was not as playful and at school she frequently was unmanageable. Physically she was normal. Mental examination showed nothing of a psychotic nature. Intellectually she was normal, as shown by psychometric tests.

The symptoms were those of phobias, compulsions and to some degree embraced arithmomania (impulse to count) and onomatomania (obsessions of a word, repeating). These may be grouped under our conception of compulsion neurosis. The various physical complaints belong to the syndrome of anxiety hysteria.

MECHANISM OF NEUROSES IN GENERAL

The first step in the understanding of obsessional or compulsion neuroses is the realization that the symptoms result from the activity of certain unconscious mental processes, the knowledge of which as given us by Freud has illuminated the entire field of the psychoneuroses. No mental phenomenon is fortuitous and each symptom is the result of a conflict between a wish which strives to find expression in consciousness and a force which represses it and even keeps back all knowledge of it. The conflict results in a compromise, and the wish comes to consciousness disguised by distortion. It then converts itself into a physical symptom (hysteria) or is transferred to an indifferent idea (obsession and compulsion). The latter method of the utilization of the affect is usually an adult mechanism.

DEVELOPMENT AND MEANING OF THIS COMPULSION

Because the patient was an only child,[3] she did not have the opportunity to give and take emotions in normal measure. Her love life of necessity was directed mainly to herself and to her father, who was the more congenial of the parents. Her adjustment in her home life was fairly adequate, but as soon as new elements were added, such as beginning school and thereby being in an environment less friendly where she also met the competition of equals and the erotic aims of other children, a readjustment became necessary. For this readjustment she was not properly prepared. She then reverted to autoeroticism, which may have been enhanced by one of the boys touching her. At first she only talked about this freely and touched her genitals without restraint. This practice she was forced to repress (by her parents), but the affect was utilized by conversion on the psychic level and the negation of her practice manifested itself. She washed her hands frequently and feared to have any one touch her which, by generalization, included also her parents. Her compulsion alarmed her parents and the ever watchful mother suppressed this manifestation. Another channel of expression became evident in physical symptoms, such as her forehead "turning." This was accomplished by displacement from below upward. That the "glass swallowing" obsession had a similar origin can be inferred from the association test. Numbers here were traceable to urination and bowel movements which, according to nursery ethics, she was taught to designate as "one" and "two," respectively. Certainly the affect displayed when she said, "Eddie goes one, two, three" can be explained only by the erotic significance with which she endowed the numerals. It is a common mechanism to convert the libido to abstract or foreign objects, such as numbers and glass. It may be suggested also that her associations for the stimulus word "Jennie" which were "one" and "papa" may be significant of the relationship which she wished to establish between herself and her father through the medium of "one" the symbolic value of which we are now aware. This she partly accomplished when she evinced that great interest in her genitals when in her father's presence, but when this was interfered with the neurosis resulted. By this means she accomplished her aim in a negative manner and also avenged her liberty of expression which was subdued by her parents. Her protest against the censorship of her pleasure became evident through the phobia that her parents might die which was the negation of a wish that they should thus be punished, an example of the omnipotence of thoughts in children and frequently found in fairy tales, where by mere wishing all may be attained.[4] In compulsive fear the wish energy is transformed into anxiety.

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[3] Brill, A. A.: Psychanalysis, Its Theories and Practical Application, Philadelphia, W. B. Saunders Company, 1914, p. 279.

Treatment was conducted along mental hygienic lines as indicated in the anamnesis and by the analysis. Six months later the parents reported that the patient had fully recovered.

CONCLUSION

Only the salient features in the analysis of this case are reported, but the few points brought out throw some light on the nature of this neurosis, which is of rare occurrence in children. This neurosis is the result of a readjustment for which the patient was not adequately prepared. The compulsion symptoms are negation acts of activities which she was forced to give up. The symptoms have as their purpose the denial of the facts, and the denial symptoms (hand washing, displacement from vulva to mouth, etc.) thus utilize the affect which needed some such disposition when once displaced from its original source.

In conclusion I want to express my thanks to Prof. Oliver S. Strong for referring the patient to me, and to Prof. L. Casamajor for the opportunity to study this case and for his helpful suggestions.

Hotel Grenoble, Fifty-Sixth Street and Seventh Avenue.

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[4] Ferenczi, S.: Contributions to Psychoanalysis, Trans. by E. Jones, Boston, R. G. Badger, 1916.

EXTRACRANIAL INJURIES OF MULTIPLE CRANIAL NERVES *

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Simultaneous lesions of the ninth, tenth and eleventh cranial nerves have frequently been observed as the result of war injuries. Often an injury of one or more additional nerves has been present (most frequently the twelfth), occasionally the cervical sympathetic and rarely the seventh nerve.

Extracranial lesions of the last four cranial nerves had rarely been observed prior to the war. Such cases were attributed either to compression by a tumor, gumma, or tuberculous gland; or to an inflammatory condition. One of the most striking examples of such a case was reported by Beck and Hassin.[1]

Lesions of the ninth, tenth and eleventh cranial nerves were more frequently noted as the result of nontraumatic causes acting about the jugular foramen. Vernet[2] has described six such cases due to compression, of which two were observed by him, and three as the result of inflammation, two of which were his own cases.

The cases presenting lesions of several or all of the last four cranial nerves have been classified on the basis of symptoms added to a pure laryngeal hemiplegia. Thus the syndrome of Avellis[3] consists of a unilateral paralysis of the soft palate, in addition to the larynx, as the result of a lesion of the pneumogastric and the internal branch of the spinal accessory nerves. The syndrome of Schmidt[4] is characterized, in addition to the foregoing symptoms, by paralysis of the sternocleidomastoid and the trapezius through the inclusion in the lesion of the external branch of the spinal accessory nerve. The syndrome of Jackson[5] includes, in addition to all of these symptoms, unilateral paralysis of the tongue resulting from a lesion of the hypoglossal nerve.

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* Read at the Forty-Sixth Annual Meeting of the American Neurological Association, New York, June 2, 1920.

1. Beck and Hassin: Med. Rec., Aug. 21, 1915.
2. Vernet: Rev. neurol. **25**:117 (Nov.-Dec.) 1918.
3. Avellis: Berl. Klinik, No. 41, 1891.
4. Schmidt: Quoted by Vernet, Footnote 2.
5. Jackson: Lancet **1**:689, 1886.

Several new groups have been added by the literature of the war. Vernet[2] has described a syndrome due to a combined lesion of the glossopharyngeal, pneumogastric and spinal accessory nerves, called by him the syndrome of the posterior lacerated foramen. Collet[6] described a combination of symptoms due to a complete lesion of the ninth, tenth, eleventh and twelfth cranial nerves under the name of glossolaryngoscapulopharyngeal hemiplegia. The same condition was described by Vernet[7] as the complete syndrome of the last four cranial nerves, and by Sicard[8] as the syndrome of the condyloposterior lacerated foramen. Villaret[9] described the syndrome of the posterior retroparatoid space, which is characterized by the addition of a lesion of the sympathetic nerve to the syndrome of the last four cranial nerves, producing thereby enophthalmos, narrowing of the palpebral fissure and myosis.

The number of syndromes is limited only by the possible combinations of complete or incomplete paralyses of these several cranial nerves, and the descriptive ability of the various observers.

One of the notable features of all the cases is that whatever other nerves might be affected the ninth, tenth and eleventh are rather consistently injured together. Such lesions are produced by wounds in the uppermost part of the lateropharyngeal space. This space is bounded above by the base of the skull in the region of the jugular foramen. The jugular foramen or posterior lacerated foramen is an opening of irregular shape and size, placed between the petrous portion of the temporal bone in front and the jugular process of the occipital bone behind. The foramen is occasionally divided into two parts by the spicules of bone which bridge it. It presents three compartments. Through the anterior compartment passes the inferior petrosal sinus; through the posterior the internal jugular vein and some meningeal branches from the occipital and ascending pharyngeal arteries; and between the two veins, in order from before backward, are the glossopharyngeal, pneumogastric and spinal accessory nerves (Fig. 1).

At its exit from the jugular foramen the jugular vein lies to the outside of the nerves; closely in front and internally lie the internal carotid artery and the accompanying sympathetic nerves (Figs. 2 and 3). At this point, Vernet states, it is possible for a projectile passing obliquely from the mastoid region on one side to the malar bone on the other to injure the three nerves and miss both the carotid and jugular vessels. This was usually the direction of the course of the

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6. Collet: Lyon méd., May, 1916, p. 121.
7. Vernet: Paris méd. **7**:78 (Jan. 27) 1917.
8. Sicard: Marseille méd., March, 1917.
9. Villaret: Paris méd., January, 1917, No. 4, p. 78.

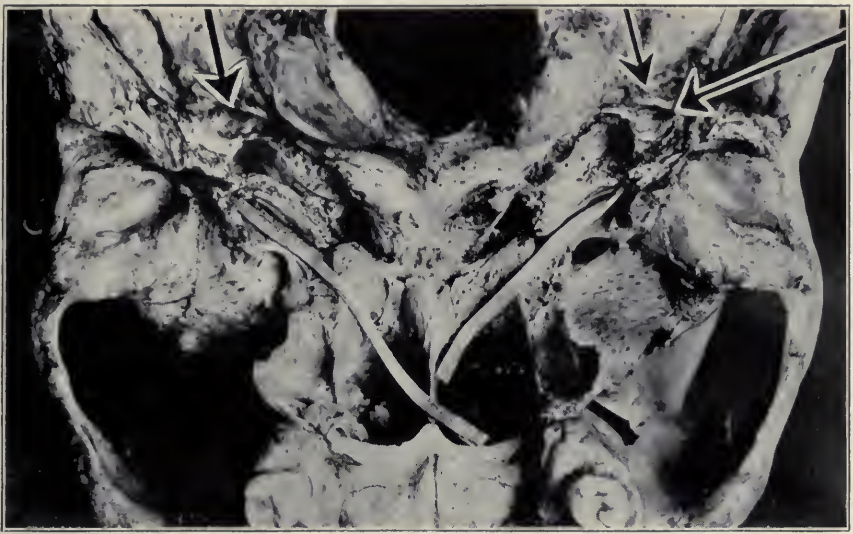

Fig. 1.—Base of skull. Arrows point to jugular fossa.

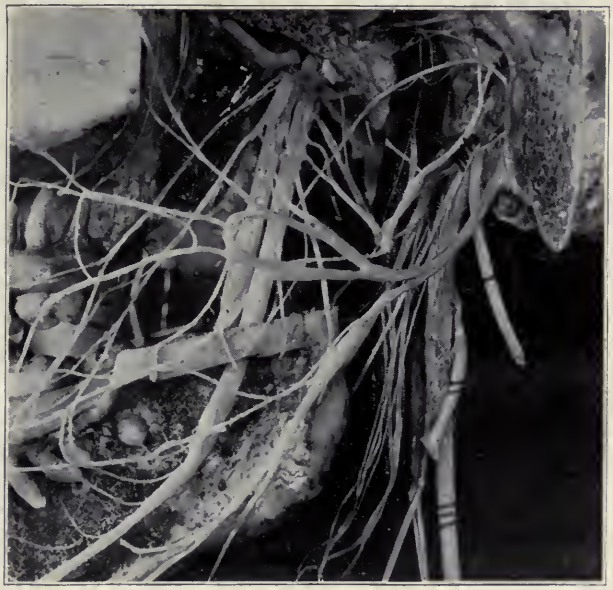

Fig. 2.—Photograph of model showing extracranial course of last four cranial nerves. The spinal portion of the spinal accessory nerve is indicated by —, the hypoglossal by =, the vagus by ≡, the glossopharyngeal by ≣.

missile in the cases under observation. In many cases, however, such a route was not followed. The fact that such patients survived the injury, which produced a paralysis of the three cranial nerves, without a contralateral hemiplegia, indicates one of two conditions: either severe injuries to the carotid and jugular vessels need not be fatal or result in a contralateral hemiplegia, or these cranial nerves may sustain severe traumatic lesions without injury of the carotid and jugular vessels. Studies of trauma of peripheral nerves have shown us that rarely does it produce anatomic section. Contusion, concussion, pressure by hematomas and arterial and arteriovenous aneurysms may likewise produce loss of function of cranial nerves.

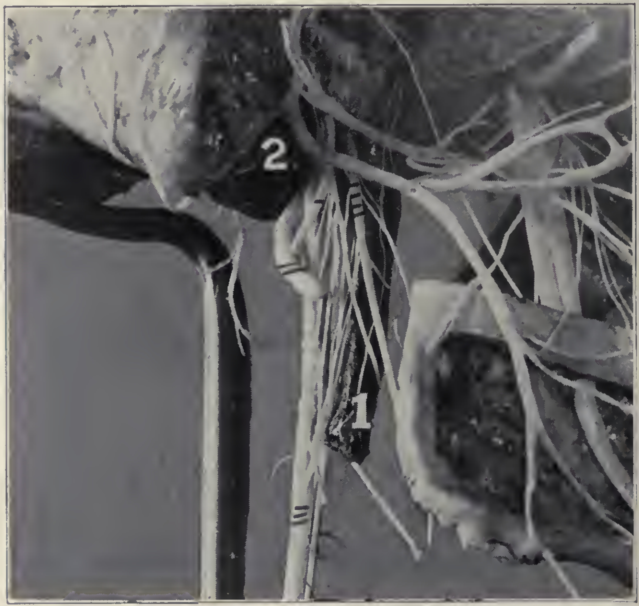

Fig. 3.—Photograph of model showing extracranial course of last four cranial nerves. The spinal accessory nerve is indicated by —, the hypoglossal by =, the vagus by ≡, the glossopharyngeal by ≣, the carotid artery by 1, the jugular vein by 2.

In certain respects, injury of the last four cranial nerves resembles injury of the brachial plexus. As a complete brachial palsy which immediately follows injury of the plexus gradually improves until only one or more of the cords are permanently paralyzed, so complete paralysis of the last four cranial nerves frequently eventuates in a permanent paralysis of only one, with partial lesion of one or more of the others.

In their peripheral course, the last four cranial nerves are in close proximity to a point a little below the level of the tip of the mastoid. Thus lesions of the ninth, tenth and eleventh cranial nerves have been caused at times by an injury not directly acting on the posterior lacerated foramen.

Whether cranial nerves other than the ninth, tenth, and eleventh are injured depends on the direction and the level of the course of the projectile. The frequent inclusion of the hypoglossal nerve in these injuries is easily understood when we recall the proximity of its exit, the anterior condyloid foramen, to the jugular foramen. Likewise it is in close proximity to the ninth, tenth and eleventh

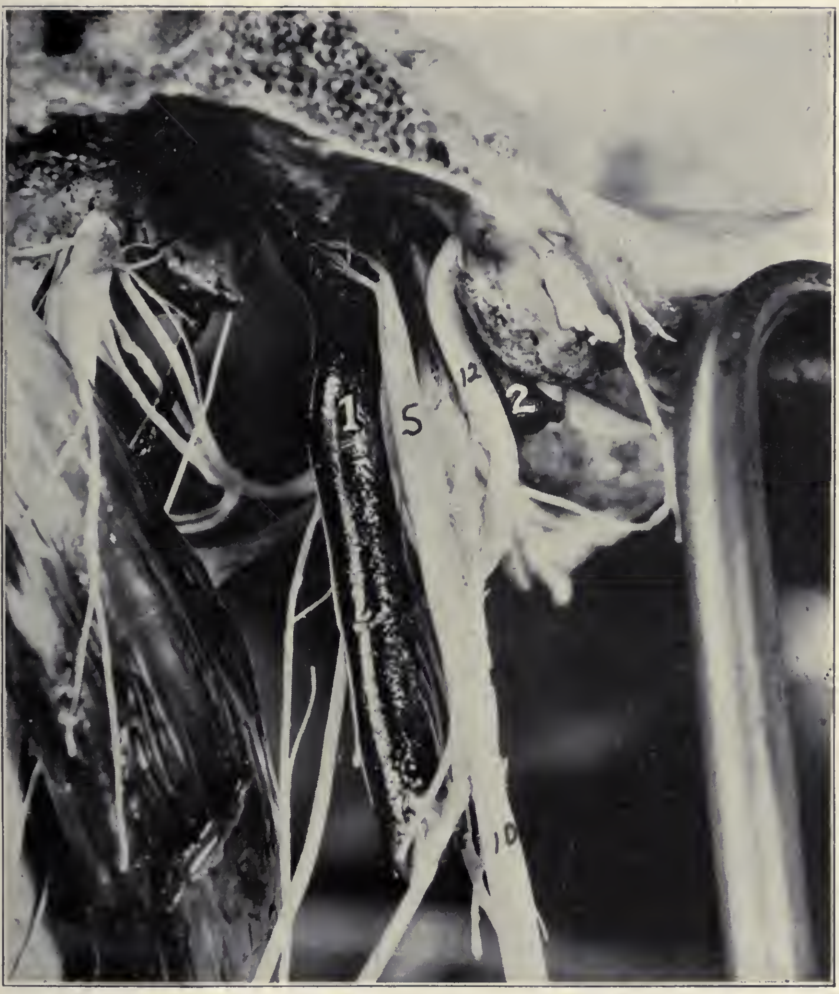

Fig. 4.—The relation of the hypoglossal (12) and sympathetic (*S*) nerves to the vagus (10); 1 indicates the carotid artery; 2, the jugular vein.

cranial nerves in the retroparotid space where its injury is frequently associated with a lesion of the sympathetic nerve. This space is described by Villaret as being bounded posteriorly by the cervical spine, internally by the pharynx, anteriorly by the internal prolongation of the parotid gland and the muscular bundle attached to the styloid process, and above by the base of the skull in the region of the jugular foramen (Fig. 4).

The symptomatology of a combined lesion of the ninth, tenth and eleventh cranial nerves is constant and easily recognized. As a characteristic triad of symptoms indicative of a complete lesion of these three nerves, Vernet proposes nasal regurgitation of fluids, dysphagia of solids and hoarseness, representing respectively paralysis of the palate, pharynx and larynx. What part of the collective symptomatology may be attributed to any one of these nerves is more difficult to interpret.

Considerable confusion exists concerning innervation of the soft palate and larynx. The specific functions of the accessory portion of the spinal accessory and the pneumogastric nerves are undetermined. Whether it would be more profitable to consider their functions together as those of the vagospinal nerves remains to be seen.

The glossopharyngeal nerve, according to Vernet, innervates the superior constrictor of the pharynx. Loss of its function results in difficulty in swallowing solids. In lesions of this nerve, the posterior wall of the pharynx deviates to the unaffected side when the patient says "ah" with the tongue pulled forward, and there is disturbance of taste in the posterior third of the tongue.

A lesion of the pneumogastric produces sensory loss on the soft palate and the posterior wall of the pharynx in addition to the well recognized disturbances of secretion (salivation) and of respiration (dyspnea or pseudo-asthma). Injury of the accessory portion of the spinal accessory nerve causes paralysis of the soft palate and larynx as well as a rapid pulse, while a lesion of the spinal portion produces paralysis of the sternocleidomastoid and trapezius muscles.

I have observed five cases of multiple lesions of the last four cranial nerves; of these the records of only three are available. Unfortunately, owing to the pressure of work and the conditions of military practice, they are not as complete as one would desire.

REPORT OF CASES

Case 1.—S. R. was wounded Aug. 10, 1918, by a machine-gun bullet, the wound of entrance being 1½ inches to the left of the fourth cervical spine, and the wound of exit half an inch below the anterior extremity of the left zygomatic arch. He was unconscious for half an hour, and when attended by a medical officer one and one-half hours following the injury he was hoarse, had difficulty in swallowing, marked salivation and dyspnea.

I examined him ten days after injury. There was a subconjunctival hemorrhage of the left eye. A hemorrhage into the mucous membrane of the posterior wall of the pharynx extended well up into the nasopharynx and down into the mouth of the esophagus. There were present a peripheral paresis of the left seventh nerve and paralysis of the ninth nerve. He had considerable difficulty in swallowing solid food. The posterior wall of the pharynx was pulled toward the right, especially when the tongue was pulled forward. No disturbance of taste could be demonstrated.

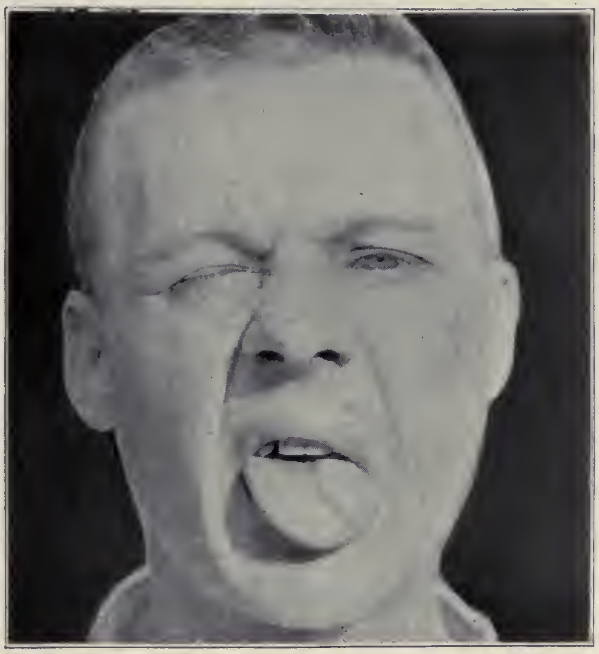

Fig. 5.—Hypoglossal palsy and difference in facial innervation.

There was paralysis of the vagospinal nerves, that is, of the accessory portion of the spinal accessory and the vagus. He had moderate salivation and sensation over the left side of the soft palate was diminished. No disturbance of respiration was noted. There was paralysis of the left side of the soft palate. The latter was pulled to the right, and in phonation this deviation was increased. There was some regurgitation of fluids. The gag reflex was absent on the left. The left vocal cord was paralyzed and the voice hoarse and nasal.

There was paralysis of the sternocleidomastoid and the trapezius. On turning the head to the right only a small portion of the sternal part of the sternocleidomastoid became prominent. The shoulder drooped. The inner angle of the scapula deviated outward, the outer angle dropped and the lower angle approximated the midline and projected under the skin. The left hypoglossal

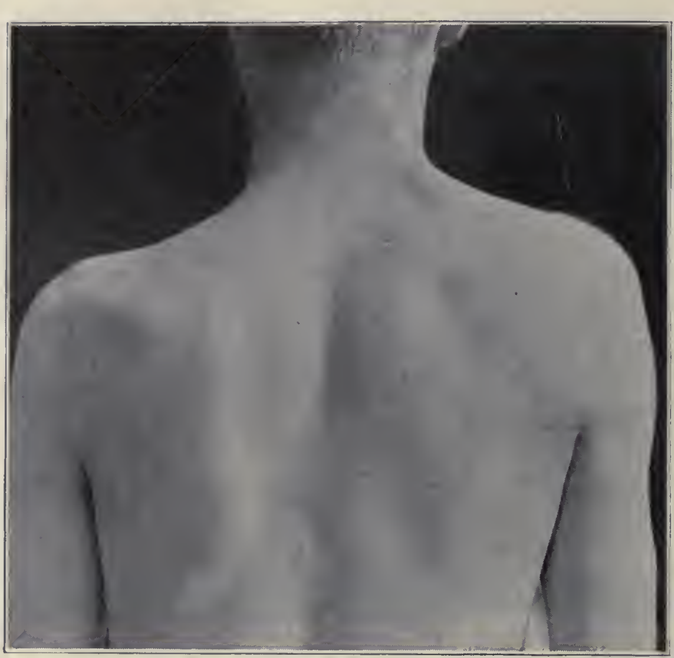

Fig. 6.—Paralysis of trapezius muscle.

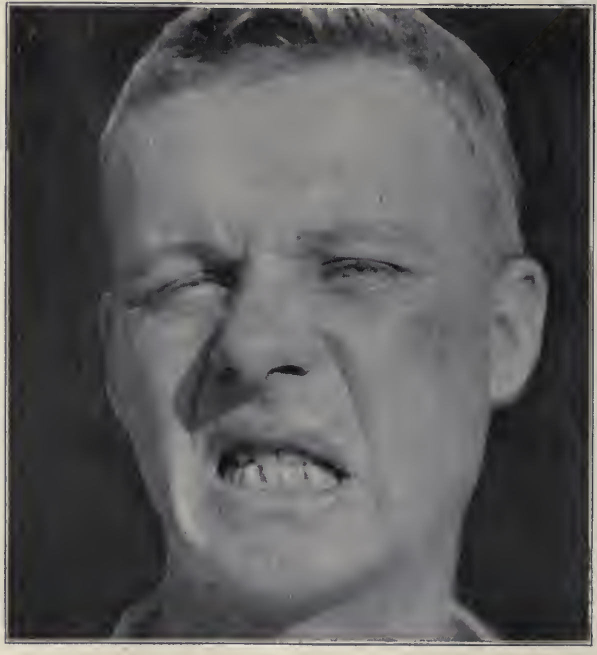

Fig. 7.—Weakness of left facial nerve.

nerve was paralyzed. The tongue, while in the buccal cavity, was pulled slightly toward the right; when it was protruded, it deviated to the left and showed atrophy and fibrillary twitching.

In this case we were dealing with an extracranial injury of the ninth, tenth, eleventh and twelfth cranial nerves and a partial lesion of the seventh. The course of the missile did not conform to the usual diagonal route from the mastoid region of one side to the malar of the other (Figs. 5, 6 and 7).

Case 2.—H. P. was wounded April 5, 1918, by a pistol bullet, the wound of entrance being just above the upper central incisors, and the wound of exit 1 inch below and 2 inches behind the right mastoid process. For several days following the injury he was unable to move his left arm and leg. He had difficulty in speaking, hoarseness, and slight regurgitation of liquids. He preferred semisolid food, as solid food was hard to swallow.

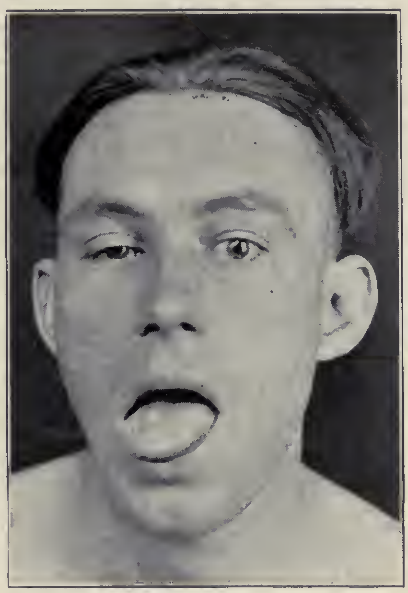

Fig. 8.—Hypoglossal palsy and narrowing of palpebral fissure, enophthalmos and myosis.

I examined him over a year after the injury. The deep reflexes on the left side were all greater than on the right; the plantars were equal and normal, the cremasteric equal, and the left upper abdominal reflex was diminished. There were no sensory disturbances over the extremities, trunk or abdomen, and no weakness in these regions was noted. It is probable that he sustained a unilateral concussion of the spinal cord at the time of injury.

A lesion of the cervical sympathetic of the left side was shown by enophthalmos, diminution in the size of the palpebral fissure and a contracted pupil. Indicating a lesion of the ninth nerve was a history of dysphagia for solids, and slight deviation of the posterior wall of the pharynx to the left on protruding the tongue. Taste was not affected.

Of the vagospinal nerves it may be said that there was a history of regurgitation of fluids and hoarseness but no history of disturbance of respiration, pulse and salivation, and no change was found.

In the distribution of the spinal portion of the spinal accessory there was found slight atrophy of the trapezius muscle, none of the sternocleidomastoid.

The twelfth nerve was paralyzed. The right half of the tongue, with the exception of the inner part of the very tip, was markedly atrophied. Slight paresis of the facial muscles of the right side was seen.

In this case we were dealing with permanent paralysis of the twelfth nerve and the cervical sympathetic, and with a dissociated and recovering lesion of the ninth, tenth and eleventh cranial nerves. In addition, a slight lesion of the seventh nerve was present. This case is illustrative of the retroparotid syndrome of Villaret. Many such cases have been noted, among others by Villaret and Faure Beaulieu,[10] Lannois, Saignon and Vernet,[11] Vidoni,[12] Halphen,[13] and Sicard and Roget[14] (Figs. 8 and 9).

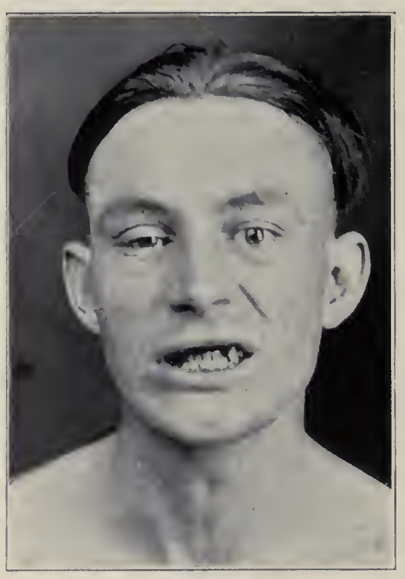

Fig. 9.—Facial nerve weakness and narrowing of palpebral fissure, enophthalmos and myosis.

Case 3.—C. L. was wounded Oct. 10, 1918, by a high explosive shell, the wound of entrance being at the tip of the left mastoid. The missile was located by roentgenogram in front of the axis in the midline. Following the injury he was unconscious for six hours.

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[10] Villaret and Faure-Beaulieu: Presse méd. **26**:591 (Nov. 21) 1918.

[11] Lannois, Saignon and Vernet: Quoted by Villaret and Faure-Beaulieu, Footnote 10.

[12] Vidoni: Quaderni di Psichiatria **4**: No. 7-8, 1917.

[13] Halphen: Reunion médicale de la IV^e Armée, June, 1917.

[14] Sicard and Roget: Marseille méd. **55**:806 (Oct.) 1918.

I examined him ten days after injury. He showed no paralysis of the extremities, trunk or abdomen. There were no sensory disturbances. No dysmetria, adiadokocinesis or other evidence of dyssynergia was found.

The findings may be divided into two groups: first, paralysis of the left facial and auditory nerves; second, paralysis of the ninth, tenth, eleventh and twelfth cranial nerves of the same side. There were complete peripheral facial palsy of the left side, loss of taste on the whole left side of the tongue and total deafness on the same side. He gave a history of tinnitus and in his record was a notation of gradually diminishing horizontal and lateral nystagmus to the right. I found such a nystagmus of mild degree. There was escape of what appeared to be cerebrospinal fluid from the left ear. Because of this, caloric tests were not performed, and turning tests were omitted because of the general condition of the patient. Roentgenograms did not reveal a basilar skull fracture.

Because the peripheral facial palsy was associated with loss of taste in the anterior two thirds of the tongue, and there was injury to both the cochlear and vestibular portions of the eighth nerve, it was concluded that the injury occurred in the peripheral part of the seventh and eighth nerves within the petrous portion of the temporal bone.

Dyspnea, hoarseness, nasal speech, palatal palsy and anesthesia, and paralysis of the left vocal cord indicated paralysis of the vagospinal nerves, and a complete or severe lesion of the ninth nerve was shown by paralysis of the superior constrictor of the pharynx and loss of taste in the posterior third of the tongue. Involvement of the spinal portion of the eleventh nerve was shown by paralysis of the sternocleidomastoid and trapezius muscles. The left side of the tongue showed fibrillary twitching.

COMMENT

The extracranial origin of the paralyses in Case 3 is not as certain as in the other two because of the involvement of the eighth nerve and the escape of cerebrospinal fluid from the ear. The absence of paralysis and sensory disturbances in the extremities and of cerebellar dysfunction speaks against a central origin. An intracranial but peripheral lesion would require an injury of wide extent, which the roentgenographic examination failed to reveal. The course of the missile was such that it traversed the upper part of the lateropharyngeal space and, on the one hand, could not have avoided injury to at least some of the last four cranial nerves, and on the other, could, and probably did, injure all of them.

The course of the missile in these cases conformed to no rule except to traverse the upper lateropharyngeal space. Whether the carotid artery or the jugular vein was injured could not be ascertained. In all of them the facial nerve was injured, as in the cases of Rimbaut and Vernet,[15] Halphen,[13] and Bourcart, Lannois and Vernet.[16] It is

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15. Rimbaut and Vernet: Marseille méd. **55**:887 (Oct.) 1918.

16. Bourcart, Lannois and Vernet: Bull. de Soc. Medico Chirurgical de la xiv, Region, Jan.-June, 1916, p. 67.

probable that many cases of injury of the last four cranial nerves might have been recorded if they had been observed soon after injury, but undoubtedly a number of them recovered, leaving permanent paralysis of only one nerve, as for example, the hypoglossal, and they were misinterpreted when examined some time after injury.

25 East Washington Street.

PUNCTURE OF THE CISTERNA MAGNA *

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A situation occasionally arises in which lumbar puncture is either impossible, impracticable or insufficient, when access to the upper reservoirs of the cerebrospinal fluid are desirable or imperative. The necessity of reaching the subarachnoid space at other points than the lumbar sac has led to such procedures as puncture in the thoracic and cervical regions, puncture through the sphenoidal fissure and intraventricular puncture. In the literature one finds no mention of cisterna puncture[1] as a clinical procedure, although it has for long been the routine method for obtaining cerebrospinal fluid in animals, and drainage of the cisterna has been employed by a number of surgeons.[2]

With other routes besides the lumbar and ventricular, the writer has had no experience, all seeming of too hazardous a nature for other than extremely rare application. Cisterna puncture, reaching as it does "the distributing center of the cerebrospinal fluid system" should, however, render access to a point of even more strategic value than thoracic, cervical or sphenoidal puncture, and should in some cases render intraventricular puncture unnecessary.

TECHNIC

The method of approach to the cisterna cerebello-medullaris (cisterna magna) was published by Wegeforth, Ayer and Essick in 1919[3] after careful preliminary study on the cadaver. In practice the procedure has been found almost always easy, and no alarming symptoms have been observed, either at the time of puncture or subsequently.

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* Read at the Forty-Sixth Annual Meeting of the American Neurological Association, New York, June, 1920.

* From the Departments of Neurology of Harvard Medical School and of Massachusetts General Hospital.

[1] Grammatical accuracy should require that we speak of "cisternal puncture" correlative with "lumbar puncture." It seems best, however, to shorten the term, using the noun instead of the adjective, and speak of this procedure as "cisterna (or cistern) puncture."

[2] Kopetzky, S. J., and Haynes, I. S.: Meningitis, Cornell Univ. Monograph, 1912.

[3] Wegeforth, P., Ayer, J. B., and Essick, C. R.: The Method of Obtaining Cerebrospinal Fluid by Puncture of the Cisterna Magna (Cistern Puncture), Am. J. Med. Sc. **157**:789, 1919.

The patient is placed on the side, as if for lumbar puncture, with neck moderately flexed. Care is taken to maintain the alinement of the vertebral column to prevent scoliosis and torsion, and in cases where comparative pressure readings are important the lumbar and cisterna needles should be on the same horizontal plane. After antiseptic preparation of the skin, usually including the shaving of a little hair, and local anesthetization with procain, the thumb of the left hand is placed on the spine of the axis, and the needle inserted in the midline just above the thumb. The needle may be pushed rapidly through the skin, but should then be cautiously and guardedly forced forward and upward in line with the external auditory meatus and glabella, until the dura is pierced.

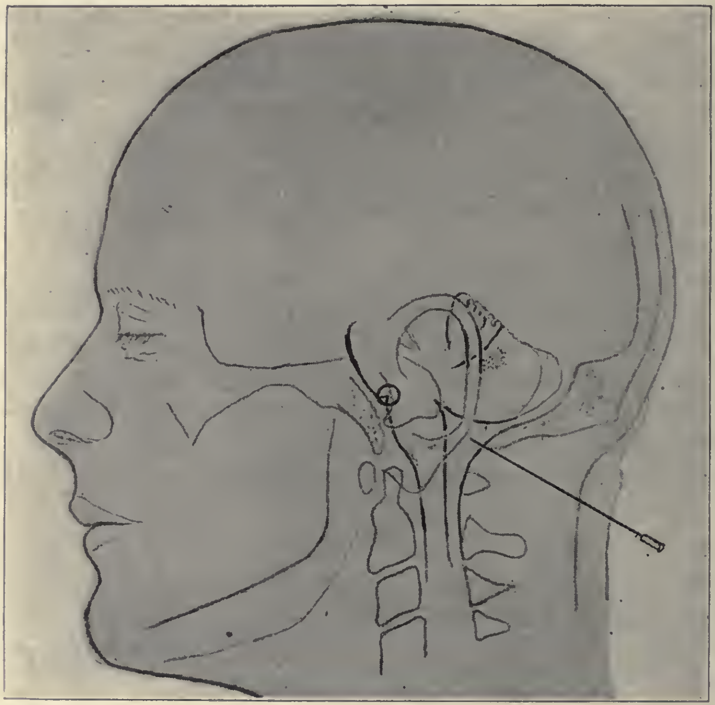

Fig. 1.—Photograph of original tracing from frozen section of head, cut in midsagittal plane. Needle in situ, one-third natural size.

If the cisterna be entered at this angle there is usually a distance of from 2.5 to 3.0 cm. between dura and medulla as shown on frozen sections; with the needle less oblique in position the distance between the walls of the cisterna becomes progressively less. Therefore, it is good practice to aim a little higher than the auditory meatus, and, if the needle strikes the occiput, to depress just enough to pass the dura at its uppermost attachment to the foramen magnum. At its entrance the same sudden "give" is felt as in lumbar puncture (Fig. 1).

The needle employed is a regular lumbar puncture needle, nickeloid, 18 gage preferred, with beveled stylet, sharp on the sides but not too sharply pointed. There is rather less variation in the depth of the tissue traversed than in the lumbar region, being in an ordinary sized adult from 4 to 5 cm., the greatest distance in the series being 6 cm. and the smallest 3.5 cm. It was found that a faint circular scratch on the needle, 6 cm. from the tip, was entirely satisfactory in judging the distance, and was preferable to the deeper markings of the Patrick needle which tend to make its insertion a little jerky and consequently less guarded.

Discomfort to the patient is usually the same as or less than in lumbar puncture, for with the wider bony opening there is less likelihood of scraping the periosteum than in passing between laminae in the lumbar region. Twice only has there been sudden transient pain on piercing the dura. Failure to reach the cisterna was experienced on two occasions, once in a patient with neck greatly retracted from acute meningitis; and once it was deemed wise to desist on obtaining no fluid at the depth of 6 cm. The after-effects of cisterna puncture have been of no consequence, even when serum has been introduced. Whether puncture headache will follow this technic is not known, as the cases in which it has been carried out were for the most part those which do not contract headache from lumbar puncture. Thus far, however, headache has not been a sequel of cisterna puncture.

The technic should not prove difficult to one familiar with lumbar puncture, especially after preliminary practice on the cadaver. It was noticeable that the apprehension attending the earlier punctures was entirely with the operator, and was not shared by the patient, whose ignorance of the proximity of the medulla served him to advantage. In spite of the simplicity of the technic it would, in the author's opinion, be unfair to the patient to perform cisterna puncture without previous experience at the necropsy table.

CLINICAL APPLICATION OF CISTERNA PUNCTURE

In the previous paper on the technic, cisterna puncture was theoretically recommended in three groups of conditions: (1) in spinal subarachnoid block following meningitis; (2) combined with lumbar puncture for irrigation of the subarachnoid space, and (3) as a route for serum injection in epidemic meningitis.

In the twenty cases in which cisterna puncture has been performed by the author are included examples of the first and third groups. Opportunity for irrigation has not yet been offered. But another field of usefulness for this procedure has been found in the early diagnosis of cord compression.

The clinical experience of the author to date rests on forty-three punctures performed in twenty cases for the following purposes:

For diagnosis or treatment of postmeningitic block in five cases; for treatment in epidemic meningitis in one case; for diagnosis and treatment of cerebral syphilis in five cases, and for diagnosis of cord compression in nine cases.

During the course of study of these cases a number of results were obtained which were apparently entirely negative and which we feel justified in taking as our normal standard. Consideration of these leads us to believe that cistern and lumbar fluids are practically identical, at least so far as protein (quantitatively determined by colorimetric method), sugar (qualitatively estimated), cells, and colloidal gold reactions are concerned. Moreover, the pressures, as registered in two manometers simultaneously, are the same, and the subarachnoid space is shown to be normally a free channel of communication as indicated by an immediate drop in pressure in either manometer when fluid is withdrawn from the other. Oscillations due to pulse and respiration, and changes in pressure from coughing, holding the breath or compression of the jugular veins are normally the same in both loci. The similarity of the fluid at these two points is a fortunate circumstance in that any difference must be explained on a pathologic basis and should be of significance.

I will proceed to the consideration of the four groups of cases for which cisterna puncture, frequently combined with lumbar puncture, was performed.

1. *For Diagnosis or Treatment of Postmeningitic Spinal Subarachnoid Block.*—Especially in neglected cases of meningitis, it is likely that adhesions will form, cutting off communication between different parts of the cerebral and spinal subarachnoid space. Favorite sites for such adhesions have been shown to be about the foramen magnum and in the thoracic meninges. In such cases of spinal meningeal block there should be a difference in the fluid obtained by cisterna and by lumbar puncture.

Five patients with possible postmeningitic block were so examined. In two this diagnosis was confirmed.

Case 1.—Baby G., 10 months old, had had convulsions since the age of 3 months, the cause of which was not apparent. In the hospital he showed unmistakable meningeal symptoms, was evidently partially blind, the optic disks being pale, and was mentally backward. The temperature ran from 99 to 101 F., and there was a moderate leukocytosis. A diagnosis of subacute meningitis was made. Lumbar puncture at one point failed to yield fluid. Puncture in another lumbar space also failed to yield fluid, but on carefully withdrawing the needle with the thumb covering the external end two drops of thin pus were found in its lumen. Cisterna puncture was then performed and 5 c.c. of slightly blood-tinged fluid was obtained under normal pressure (90 mm. of water). The fluid was found to be normal in protein and cells, except for the slight amount of blood present, and free from organisms. Unfortunately no culture was made from the lumbar pus. A diagnosis of spinal subarachnoid block was made, with probable persistent infection of the spinal meninges, but with evidence against residual infection of the cerebral meninges at the base (ventricular infection, localized cortical meningitis and brain abscesses were of course not excluded).

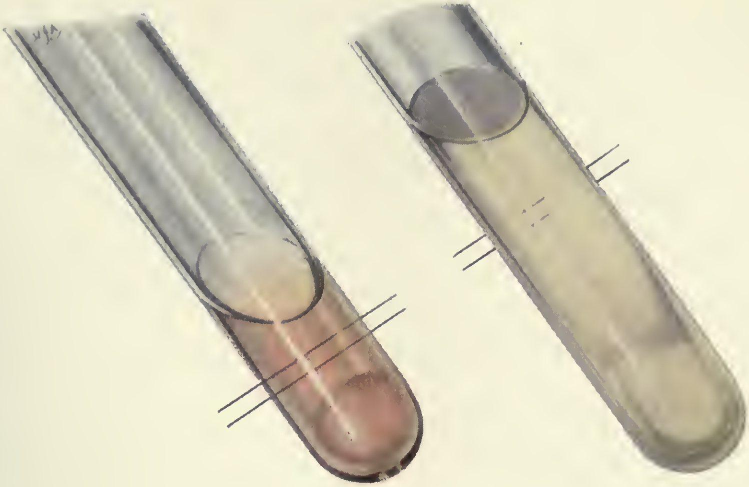

Fig. 2 (Case 2).—Cistern fluid (right) purulent; Staphylococcus aureus. Lumbar fluid (left) clear, showing Froin syndrome of xanthochromia and massive coagulation; no organisms. From a case of postmeningitic spinal subarachnoid block.

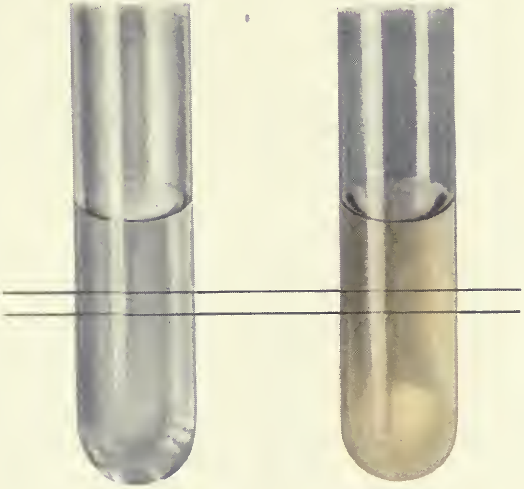

Fig. 3.—Cistern fluid (left) normal. Lumbar fluid (right) Froin syndrome. From a case of compression of spinal cord by dislocation of thoracic vertebra.

CASE 2.—Mr. S. was admitted to the Cambridge Hospital, Sept. 20, 1919, on the service of Dr. F. R. Jouett and Dr. J. P. Nelligan, to whom I am indebted for seeing this patient. He had been sick about three weeks and although mentally clear presented unmistakable signs of acute meningitis, with marked head retraction and a positive Kernig reaction, and a temperature of 102 F. He also presented a spastic paraplegia with some atrophy of the legs and thighs, indicative of a diffuse myelitic process. Lumbar puncture yielded only 7 c.c. of clear, yellow fluid which promptly clotted solidly and in which no organisms could be demonstrated on film or culture, and only a rare cell could be found. In fact, the lumbar fluid was typical of the "syndrome of Froin." Cisterna puncture yielded an abundant yellowish white purulent fluid, under high pressure (400+ mm.) containing a large number of cocci, identified on culture as *Staphylococcus aureus* (Fig. 2). The cisterna was washed out with physiologic sodium chlorid solution. On a subsequent occasion the patient's own serum was introduced with the hope that it might contain curative substances. The temperature became normal, and symptoms of acute meningeal irritation gradually subsided; some days later a third cisterna puncture yielded an almost clear fluid, showing few cells and no organisms. On account of persistent high leukocytosis in the blood and a slight degree of optic neuritis it was thought that infection of the ventricles might be present; to settle this point combined cisterna and ventricle punctures were performed; both of these spaces proved to be free from organisms and pus; and incidentally permeability of the velum medullare was established by the ready passage of serum from the ventricle into the cisterna magna. Clinically, this patient began to improve immediately after the first cisterna puncture, the signs of active meningitis rapidly disappearing. The spinal subarachnoid block, however, remained, as shown by persistence of signs of myelitis and inability to obtain more fluid from the lumbar sac.

By means of combined cisterna and lumbar punctures we were able in the first case to demonstrate persistent pus in the spinal meninges, the basal cerebral membranes being presumably free from infection; in the second to demonstrate persistent acute staphylococcus meningitis in the cerebral meninges with spinal meningeal block, and to treat successfully the cerebral infection by way of the cisterna magna.

2. *For Early Treatment in Meningococcus Meningitis.*—Serum treatment has reduced the mortality of epidemic meningitis to approxmately 25 per cent. Why do we continue to lose even one in four cases? A number of reasons may be adduced: The use of a serum that is inactive in a given lethal case; the presence of an organism of especial virulence; the establishment of treatment too late. But a most important reason seems to be that the serum does not reach the earliest pathologic lesions in sufficient concentration. While the exact method and points of entrance of the meningococcus to the meninges are not yet certain there is both clinical and experimental evidence [4] pointing

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[4] Weed, L., Wegeforth, P., Ayer, J. B., and Felton, L. D.: A Study of Experimental Meningitis, Rockefeller Institute, Monograph Series No. 12, 1920.

toward the cerebral rather than the spinal meninges as the earliest infected. Substances introduced in moderate amounts and under normal pressure into the lumbar subarachnoid space have been repeatedly shown to reach little higher than the base of the brain, and in this series I have been able to confirm this fact, as much as 20 c.c. of serum introduced into the lumbar sac not appearing at the cistern during the period of injection. Similarly, injections of 30 c.c. of India ink into the lumbar sac of a cadaver barely blackens the base of the brain; but when a similar amount is placed in the cisterna magna, not only is the base well blackened, but also the cerebral cortex on both sides. Theoretically then, antimeningococcus serum should be most efficacious in early meningitis if given by the cisterna route. Only one patient has been so treated by me and that one not only by the cistern but also by the lumbar and venous routes. In this patient, however, ventricular infection resulted and it was not until the disease was attacked by the ventricular route also that recovery took place. Cisterna injections were employed only three times in this case. It was one of my earliest uses of this method and I admit that my inexperience with the technic, in a case made difficult by head retraction, prevented a persistence which I should not again countenance. The case is mentioned as showing the possibility of administering serum in epidemic meningitis by the cisterna route, which on theoretical grounds might well be the method of choice.

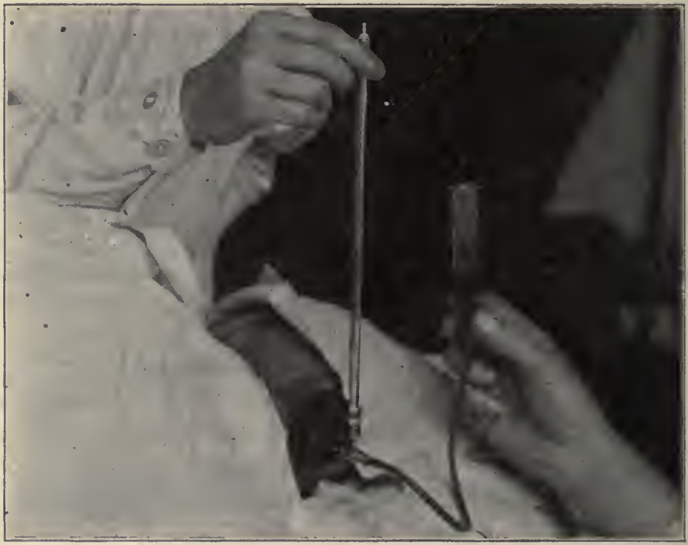

Fig. 4.—Method of administering serum by cisterna puncture.

3. *For Diagnosis and Treatment of Syphilis of the Nervous System.*—In four cases of late cerebral syphilis arsphenamized serum (Swift-Ellis technic) has been used. A fifth patient was punctured for diagnosis alone. As much as 20 c.c. of undiluted serum has been employed with no disturbance to the patient at the time of puncture or subsequently. The technic is similar to that used in lumbar injections, special care being taken to alter pressures very slowly (Fig. 4).

Two of the patients treated in this manner suffered from progressive optic atrophy (so-called "primary"), one had bilateral facial pain, resistant to other forms of antisyphilitic treatment, the fourth was a case of general paresis. Needless to say, in all cases the laboratory tests were confirmatory of the diagnoses.

This is not the place to discuss the merits and demerits of serum therapy of neurosyphilis; nor do I believe that any deductions as to the prognosis in any of these cases is justified at this time. However, it may fairly be said that the results to date are sufficiently encouraging to recommend further trial. It may also be definitely stated that in these twenty-one injections in four patients the procedure has been easy of application and somewhat more comfortable for the patient than lumbar injections, and that this technic unquestionably places the serum in greatest concentration in the cerebral meninges.

4. *For Diagnosis of Cord Compression Other than Cases of Postmeningitic Block.*—It is assumed as proved that the cerebrospinal fluid obtained from the lumbar sac passes downward from the cerebral reservoirs of which the cisterna magna is the greatest. We have known for some years that obstruction to this downward flow other than by postmeningitic adhesions will cause certain changes in the fluid below the level of cord compression (syndromes of Froin and Nonne). Moreover, we know from the results of a few operations and from experimental cord compression[5] that the fluids above and below the level of compression are different. These facts, already recognized, led us to use combined cistern and lumbar punctures in the diagnosis of spinal subarachnoid block, in the hope that differences, even slight, in the two fluids would give us information of value. This hope has been realized.

The criteria on which our judgment in such cases is based are two-fold: (1) evidence of mechanical obstruction to the free passage of fluid in the spinal subarachnoid space, as indicated by (*a*) pressure relations in the two loci, especially changes in pressures which result after withdrawal of fluid (Fig. 5), (*b*) variations in the normal oscillatory movements of the fluid, dependent on the cerebral pulse and respiration, and (*c*) gross changes in fluid pressure caused by compression of the jugular veins, and by coughing, all indicative of mechanical interference with the continuity of the spinal fluid column; (2) evidence of different chemical composition of the two fluids, as shown by comparative protein determinations, variations from the normal supposedly due to alterations in the meninges, allowing increased transudation of protein into the spinal fluid.

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[5] Ayer, J. B.: Cerebrospinal Fluid in Experimental Compression of the Spinal Cord, Arch. Neurol. & Psychiat. **2**:158, 1919.

While it is certain that tumors and other lesions which compress the spinal cord frequently cause isolated protein increase in the spinal fluid below the point of pressure, it is possible that certain degenerative diseases of the cord may produce similar changes. In the differential interpretation of these two groups of cases presenting similar pathologic fluids it is hoped that the comparison of two fluids will be of greater value than the examination of the lumbar fluid alone.

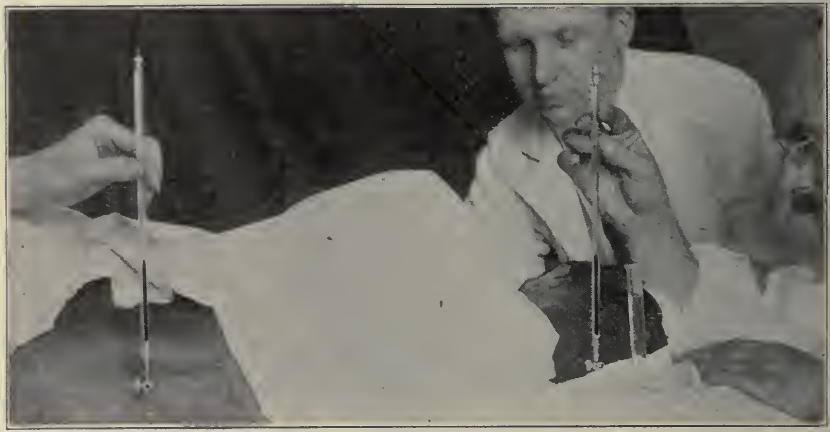

Fig. 5.—Method of combined lumbar and cisterna puncture, with manometers for measuring pressure. Ten c.c. of fluid has been released from the cisterna with equal drop in pressure in both manometers. Normal. The oscillatory movements, with pulse and respiration, and gross changes of pressure on coughing and on compression of the jugular veins are normally equal in the two manometers.

Nine cases, suspected of spinal block other than postmeningitic, have been thus examined. In three, there was no evidence of block, as indicated by mechanical continuity of the subarachnoid space and similar chemical findings in the two fluids. A third gave very slight difference in the fluids and judgment is suspended in interpreting its significance. Five cases showed unmistakable difference in the fluid findings, and three of these have thus far come to operation and the cause of obstruction has been found.

Two examples, showing different degrees of subarachnoid block, are given.

Case 3.—Mr. J. G., aged 24 years, was admitted to Massachusetts General Hospital, April 7, 1920, with a history of illness, beginning with pain in the chest five months before. A few weeks later he noticed increasing weakness of his right leg and numbness in both legs. The numbness and weakness had progressed to such an extent that walking had become greatly affected. There was also difficulty in starting urine. Examination showed partial Brown-Séquard paralysis with an indefinite zone of hyperesthesia at the level of the tenth thoracic segment.

Comparative cisterna and lumbar punctures, April 9, yielded findings summarized in Table 1.

TABLE 1.—Comparison of Findings in Cerebrospinal Fluid from Cisterna and Lumbar Punctures

	Cisterna	Lumbar
Pressure, in mm., initial *	150	170
After withdrawal of 5 c.c. from cistern **	130	165
After withdrawal of 5 c.c. from lumbar	135	85
After withdrawal of 10 c.c. from lumbar	135	30
After waiting five minutes	170	30
Character	Clear, Colorless, No Clot	Clear, Colorless, No Clot
Protein (alcohol precipitation)	Slightly +	++++
Total mg. per 100 c.c. (sulphosalicylic precipitation †)	83	1112
Globulin (ammonium sulphate precipitation)	0	++
Colloidal gold test	±±±0000000	1223332000
Erythrocytes and leukocytes	0	0
Wassermann reaction		—

* Oscillations of fluid with pulse, respiration and on coughing normal and equal.
** Oscillations remain normal and equal in both manometers.
† Unpublished method.

By this examination it is evident that there is obstruction to the free passage of fluid in the spinal subarachnoid space, as shown by manometer readings after withdrawal of fluid (Fig. 6). The examination of the fluids likewise shows a marked chemical difference, the cistern fluid appearing almost normal, the lumbar fluid presenting the "Nonne syndrome" (Figure 7).

Laminectomy was performed by Dr. W. J. Mixter and a cholesteatomatous cyst of the pia was evacuated. Subsequently the patient improved, and, May 12, combined punctures were repeated. The findings are summarized in Table 2.

TABLE 2.—Comparison of Findings in Cerebrospinal Fluid from Cisterna and Lumbar Punctures

	Cisterna	Lumbar
Pressure, in mm., initial *	210	180
After withdrawal of 10 c.c. from cistern	160	130
After withdrawal of 10 c.c. from lumbar	100	70
Character	Clear. Colorless, No Clot	Clear, Colorless, No Clot
Protein (alcohol precipitation)	Slightly +	+
Total mg. per 100 c.c. (sulphosalicylic acid precipitation)	166	192
Globulin (ammonium sulphate precipitation)	0	0
Colloidal gold test	±±±0000000	++++++0000
Leukocytes	2	0

* The fluid levels were the same. The head was 3 cm. lower than the lumbar spine.

The second examination showed a free subarachnoid space (Fig. 8) and the two fluids were almost normal. In this patient with a known

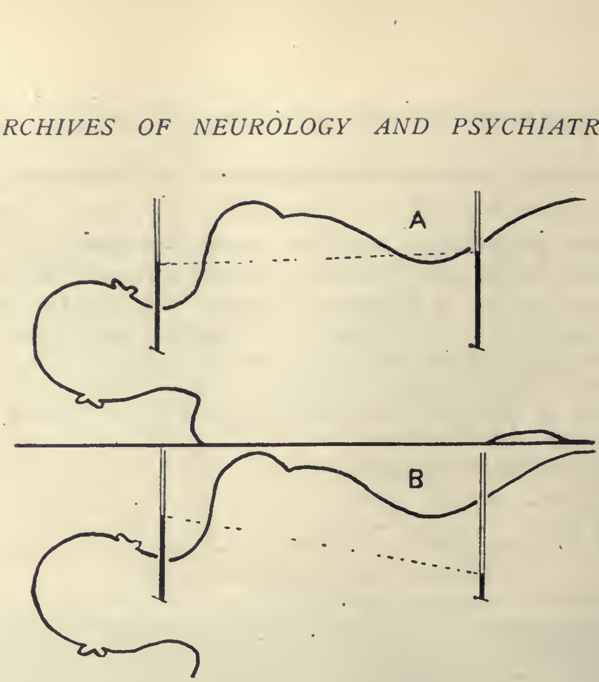

Fig. 6.—(Case 3).—Before operation. Compression of spinal cord by cholesteatomatous cyst. *A,* initial pressure in cistern and lumbar sac nearly equal. *B,* after withdrawal of fluid from the latter, the pressure below the point of compression falls, and above this point it remains elevated.

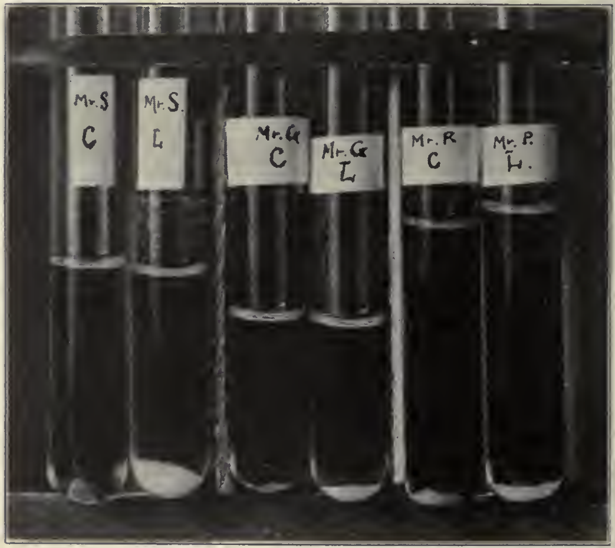

Fig. 7.—Comparative fluids from cistern, *C,* and lumbar puncture, *L,* from three cases of known compression of the spinal cord. In each case protein has been precipitated by alcohol from equal amounts of spinal fluid, the lumbar fluid in each showing the greater amount. Mr. S., tuberculosis of upper thoracic spine. Mr. G. (Case 3), cholesteatomatous cyst of pia, lower thoracic region. Mr. P. (Case 4), vertebral degenerated myeloma, lower thoracic region.

pathologic block of the spinal subarachnoid space, we were able before operation to demonstrate not only a chemical difference in the two fluids, but also mechanical obstruction to the flow of the spinal fluid. One month after surgical relief and coincident with clinical improvement, the two fluids appeared almost similar, and conclusive evidence was obtained that reestablishment of the spinal subarachnoid space had been brought about.

Case 4.—Mr. P. T. P., 33 years of age, in December, 1919, complained of abdominal pain, which was referred to the spine and shoulders, and subsequently localized in the abdomen, which became greatly distended. December 26, while he was lying down, both feet became numb, the numbness ascending within a few hours to the hips, accompanied by complete bilateral paralysis of the lower extremities, retention of urine and incontinence of feces. Examination in March showed complete paraplegia and anesthesia up to the eleventh thoracic vertebra on the left side and the ninth thoracic vertebra on the right. While no voluntary motion was possible, reflexes of defense were conspicuous. A roentgenogram of the thoracic spine was negative.

March 17, combined cisterna and lumbar punctures gave results which are summarized in Table 3.

TABLE 3.—Comparison of Findings in Cerebrospinal Fluid from Cisterna and Lumbar Punctures

	Cisterna	Lumbar
Pressure, in mm., initial *	120	120
After withdrawal of 5 c.c. from cistern	90	90
After withdrawal of 5 c.c. from lumbar	80	80
After withdrawal of 3 c.c. more from cistern	60	60
Character	Clear, Colorless, No Clot	Clear, Colorless, No Clot
Protein (alcohol precipitation)	Slightly +	Greatly +
Total mg. per 100 c.c. (sulphosalicylic acid precipitation)	100	250
Globulin	0	+
Colloidal gold test	1111000000	1543222100
Erythrocytes and leukocytes	0	0
Sugar	+	+
Wassermann reaction		—

* Oscillations with pulse, respiration and cough normal and equal in both fluids.

These findings show no mechanical obstruction to the downward passage of the spinal fluid, but marked discrepancy is seen in the protein content in the two fluids indicative of increased transudation into the fluid from a source between the two needles.

On account of the rapid onset of the symptoms of transverse myelitis the patient was sent home with a diagnosis of myelitis, probably degenerative. The next month he returned, laminectomy was performed, and an infected vertebral myeloma, pressing on the dura, was found.

While we must admit the coincidence of both compression and a toxic agent in the causation of the symptoms in this case, it is the opinion of the author that the spinal fluid changes are principally the result of the compression.

The third case in which the diagnosis of cord compression was demonstrated at operation presented both mechanical and chemical changes in the fluids even more striking than in Case 3. The pathologic lesion in this case was tuberculosis of the upper thoracic spine.

On the basis of the clinical examination in these three cases the neurologist might say that the diagnosis of cord compression was already sufficient without any help from spinal fluid findings. This was certainly true of the man with Pott's disease; the diagnosis was almost assured in Case 3, but not at all certain in Case 4. In fact, this last patient was sent home after two weeks' study in the hospital with a tentative diagnosis of degenerative myelitis. Every diagnostician will admit of finding cord degeneration at operation when tumor has been expected, and, vice versa, a tumor or cyst or other cause of compression is not infrequently present when least expected. Confirmatory evidence from spinal fluid examination should therefore be most welcome in all such cases. That negative findings are proof against the presence of cord compression is not maintained, for twice I have obtained normal fluids from below tumors (lumbar puncture alone); that positive spinal fluid findings are usually found below the level of cord compression has been my experience and that of many writers. It is to be hoped that a comparison between the fluid findings above and below a compressed area of the spinal cord, made possible by the method here described, will give information of greater significance and reliability than is to be obtained from examination of the lumbar fluid alone, and thus the procedure will aid in earlier diagnosis of spinal cord compression.

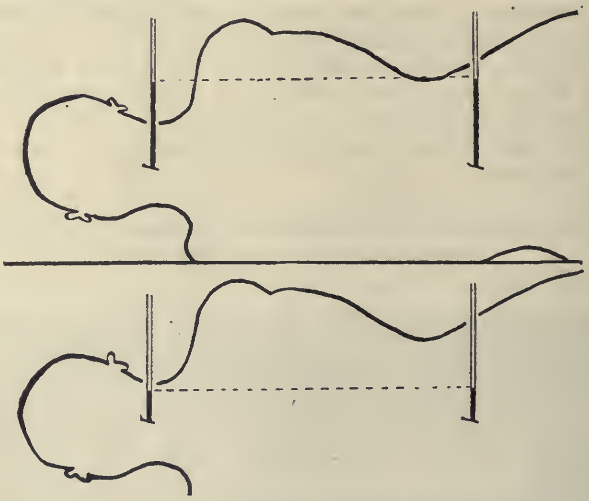

Fig. 8. (Case 3).—After operation. Pressure in cistern and in lumbar sac falls promptly and equally whether fluid is withdrawn from one or the other needle. The continuity of the subarachnoid space has been reestablished by operation.

CONCLUSIONS

Puncture of the cisterna magna should be employed when it is desirable to reach the upper reservoirs of the cerebrospinal fluid system. It may be employed alone but is frequently used to greater advantage in connection with lumbar puncture, and occasionally with ventricular puncture.

The technic has been found to be easy to acquire, and should prove safe in the hands of a careful operator. Preliminary experience on the cadaver should, however, be insisted on before its application.

This procedure, as carried out forty-three times in twenty patients, has proved of value either for diagnostic or therapeutic purposes. It has been used to advantage in the diagnosis and treatment of cases of postmeningitic subarachnoid block. It has been employed in the treatment of a selected group of cases of cerebral syphilis and in one case of epidemic meningitis. In connection with lumbar puncture it has proved of value in the diagnosis of cord compression from causes other than meningitis.

THE COINCIDENCE OF CERVICAL RIBS AND SYRINGOMYELIA *

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The Berlin surgeon Borchardt[1] appears first to have observed the coexistence of cervical ribs and syringomyelia, in a woman 35 years old, who also was examined by Oppenheim. The latter[2] in his textbook comments on this case and points out that cervical ribs belong to the so-called stigmas of degeneration and may be combined with other disorders which are not caused by the ribs but, like them, are features of an underlying neuropathic diathesis. He specifically mentions hysteria, hypochondriasis and spinal gliosis. In connection with the latter he refers to the case mentioned above and to one reported by Marburg.[3] This case, which occurred in a woman 19 years old, was complicated by scoliosis which caused the cervical ribs to be prominent and to be mistaken for a bony tumor until operative removal was attempted. Later the appearance of sensory dissociation, painless burns, muscular atrophy and lagophthalmos led to a positive diagnosis of syringomyelia. I have found no other cases in a rather hasty search of the literature, but the combination has been the subject of comment. Thus, Haenel[4] warns that the onset of brachial pains, atrophies and cervical scoliosis or kyphosis in a young person with cervical ribs should lead one to suspect syringomyelia. Jelliffe and White[5] state: "Several cases have been observed in which a cervical rib was removed from a patient suffering from syringomyelia to which the symptoms were due."

In Streissler's[6] comprehensive review of the subject of cervical ribs, Oppenheim's idea of their being degenerative stigmas is looked on with favor. He mentions scoliosis in the cervical and upper thoracic region as a frequent association and does not consider it secondary to the rib, also spina bifida and other malformations, such as harelip,

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* Read at the Forty-Sixth Annual Meeting of the American Neurological Association, New York, June, 1920.

[1] Borchardt, M.: Symptomatologie und Therapie der Halsrippen, Berl. klin. Wchnschr. **38**:1265, 1901.

[2] Oppenheim, H.: Lehrbuch der Nervenkrankheiten, Ed. 5, pp. 498 and 440.

[3] Marburg, O.: Syringomyelie und Halsrippe, Wien. klin. Rundschau **20**: 241, 1906.

[4] Haenel, H.: Handbuch der Neurologie **2**: 609.

[5] Jelliffe and White: Diseases of the Nervous System, Ed. 1, p. 301.

[6] Streissler, E.: Die Halsrippen, Ergebn. d. Chir. u. Orthop. **5**:280, 1913.

cryptorchidism, dislocated lenses, clubfoot, congenital lipoma; finally, combination with psychoneuroses, syringomyelia, multiple sclerosis (Levi),[7] muscular atrophy (Spiller and Gittings).[8]

REPORT OF CASES

CASE 1.—*History.*—The early history of this case is related by the late Dr. John B. Murphy.[9] A man, 34 years of age, was admitted to Mercy Hospital, Nov. 2, 1915. Nine months before, on Feb. 13, 1915, he slipped on the ice and fell. Two days later he had fever (100 F.), pain in the cervical region of his spine, and universal muscular tenderness, and took to bed for two days under the impression that he had grip.

Ten days after the onset of the pain in the cervical region of the spine, it radiated down the left upper extremity until it reached the fingers, where it remained. The pain was worse during the night. There was, however, no atrophy of the muscles of the hand and no alteration in the color of the skin. Pain was present in the right upper extremity.

Examination.—Palpation of the neck revealed an elongation at the tip of the transverse process of the left seventh cervical vertebra—at the site which appears to be just under the point of greatest pain irritation; the space between the process and the clavicle was diminished but not occluded. In making the comparative examination between the right and left sides it was found that on the right side the whole thumb could be inserted into the supraclavicular space, while on the left but two thirds of the digit could be inserted. There was a seventh cervical rib on the left side which put tension on the eighth cervical nerve on its way to the ulnar.

The cervical rib was plainly demonstrated by the roentgen ray.

Treatment and Course.—On Nov. 5, 1915, Dr. Murphy operated and during the operation he said: "Now you can see in the field the anterior portion of the cervical rib; instead of being a round, blunt process, it is articulated and exhibits a sharp dentate edge which nips the brachial plexus and accounts for the pain radiating down along the arm to the forearm and hand. The plexus has been displaced." After removing the rib he added: "With the rib out of the way, it is clearly to be seen where the brachial plexus was compressed by the rib. The nerve to the rhomboids was involved, showing that the fifth cervical nerve from which this branch takes origin, felt the stress of the compressing rib. The knife-blade-like edge of the cervical rib had irritated the brachial plexus until a membrane formed upon one of its constituents—the eighth cervical from which the ulnar nerve is derived."

A postscript dated Jan. 7, 1916, says: "The patient made an uneventful recovery, and at the present time the preoperative thickening which was present in the lower portion of the left side of the neck has to a great extent subsided."

This apparently happy ending was really only the introductory chapter to the story of this patient's troubles. He soon began to have severe pain in the scar and then in the whole arm as before. Examination by the writer on

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[7] Levi, H.: Beitrag zur Kasuistik. der Halsrippen, Neurol. Centralbl. **23**: 988, 1904.

[8] Spiller, W. G., and Gittings, J. C.: Progressive Muscular Atrophy of Cervicobulbar Type, Occurring with Cervical Rib, New York M. J. **84**:683, 1906.

[9] Murphy, John B.: Cervical Rib, The Clinics of John B. Murphy **5**:227 (April) 1916.

Oct. 6, 1916, revealed analgesic areas along the outside of the left arm and forearm and absence of the wrist and elbow reflexes on the left side while these reflexes were normal on the right side.

A more detailed examination was made on March 31, 1917: There were relative analgesia and thermo-anesthesia on the left side in the distribution of all the segments from the second cervical to the third thoracic. Circumference of the arms: right, 27 cm.; left, 23.5 cm.; forearm, right, 23 cm.; left, 21.5 cm.; hand, right, 20 cm.; left, 18 cm.

A lumbar puncture was made at this time and the Wassermann, Lange, and Nonne tests were negative; the cell count was 7.

A tentative diagnosis of syringomyelia was made and the patient was given roentgen-ray treatments of the lower cervical region of the spine.

Since that time the patient has been examined every few months. Both the subjective symptoms and the findings have remained practically the same. He was recently thoroughly studied by Dr. William G. Spiller, who also made a diagnosis of syringomyelia.

Case 2.—*History.*—A woman, about 40 years old, was referred to the writer for examination by Dr. George F. Dick in October, 1916. Thirteen years previously she had first noticed atrophy in the right hand, and in the left hand during the preceding year only. There was no pain but the hands had become weak. Roentgen-ray examination had revealed distinct cervical ribs on both sides. An operation had been considered but on account of the high degree of atrophy and disability of the hands a more serious disorder was suspected.

Examination.—Examination at this time, in addition to the atrophy of the small muscles of the hands, revealed extensive analgesia and thermal anesthesia of both arms and of the chest and back on both sides down to the fourth rib. The right pupil was larger than the left and the right palpebral fissure smaller than the left, showing involvement of the cervical sympathetic. The tendon reflexes in the legs were moderately increased but there was no clonus and the plantar reflexes were normal.

The patient was examined again in April, 1920, and the picture of syringomyelia was more fully developed. The atrophy of the hands was marked. The thumbs and fingers were hyperextended at the metacarpophalangeal joints and flexed at the phalangeal joints. There was no power of abduction and adduction of the fingers or of opposition of the thumbs. Flexion at the wrists was weak; extension a little better. The other arm movements were quite strong. The loss of pain and temperature sense now involved the neck and the trunk as far down as the level of the umbilicus. Tactile sensation remained normal. The knee reflexes were increased and ankle clonus was present on both sides. The abdominal, wrist and elbow reflexes were absent, the plantar reflexes normal.

Case 3.—*History.*—For the privilege of seeing and recording this case I am indebted to Dr. Lewis J. Pollock, who already had made a diagnosis of syringomyelia. He and I had examined the first patient reported in this paper a short time before and with the coincidence of the two affections in mind Dr. Pollock had sent the patient to Dr. Hollis E. Potter for roentgenologic examination which, to the great surprise of all concerned, showed bilateral cervical ribs, much larger on the right side.

The main points in the clinical history, which did not at all suggest cervical rib were:

The patient, a business man, single, 32 years old, when examined in July, 1919, a few days after excessive sexual indulgence in 1907, noticed a sensation of warmth in the right leg. During the following month the whole right side became involved and there was a sensation of tingling. The right side became somewhat rigid. A few days after the onset a physician had discovered analgesia and thermal anesthesia of the whole right side, while tactile sense was preserved. At the time of examination the chief complaint was numbness in the right leg.

Examination.—Examination revealed loss of temperature sense in the whole right lower extremity and diminution of this sensation on the right side of the rest of the body, gradually diminishing upward; even the face was slightly involved. Sensation to pain was diminished in the right leg and slightly so in the right arm and right side of the trunk. Tactile and joint sensation were normal and vibration sense slightly diminished on the left side. All of the tendon reflexes, as well as the abdominal and cremasteric reflexes, were diminished on the right side, the ankle reflex showing the greatest degree of diminution. There was no atrophy or paralysis, and coordination was normal. The Wassermann test with blood and spinal fluid and all other spinal fluid tests were negative.

CONCLUSION

In conclusion, I wish to state that were I to be guided by my own experience, which is very limited, I would be inclined to regard all bearers of cervical ribs with extreme suspicion and to accept them as possessed of well-balanced minds and structurally normal central nervous systems only after very close scrutiny. Three patients whose local symptoms led me to advise operation, which in each case was a surgical success, were made decidedly worse, one passing through a severe postoperative psychoneurosis, the other two having now led for years miserable lives as profound hypochondriacs with persistence of the local symptoms in spite of repeated subsequent exposures of the brachial plexus and injections of procain and dilute alcohol.

Fortunately, this gloomy picture is incorrect when a broad view of the situation is taken. Good results from operation in a large series of cases are reported by so many reliable observers that even repeated ill luck in one person's experience should not shake our optimism.

30 North Michigan Boulevard.

THE FREQUENCY OF ALBUMINURIA WITH CASTS IN EPILEPTICS FOLLOWING CONVULSIVE SEIZURES.

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It has frequently been stated that in epileptic individuals, albumin with casts is at times found in the urine voided immediately after attacks. Its presence is accounted for by several observers as probably due to admixture of semen liberated into the urine by pressure on the seminal vesicles during the convulsive movements. By others it is attributed to muscular contractions. In the first instance the condition would fall in the class of accidental albuminuria, in the second it might come under the heading of physiologic albuminuria. Textbooks on clinical pathology and diagnosis discuss the condition under neurotic albuminuria.

As to the frequency of abnormalities in postseizure urine in epileptics, Simon [1] states: "in a number of cases in which I had occasion to examine urine voided after an attack (epileptic), albumin was usually absent." In another place the same author states that the cases referred to were those exhibiting the petit mal attacks, and that one case which had been in the epileptic state for forty-eight hours showed albumin, semen being absent. Nothnagel [2] could not demonstrate any regularity in the occurrence of albuminuria in a number of cases of epilepsy under his observation, neither was he able to correlate the positive findings in some cases with the nature of the attack—grand mal or petit mal. Munson,[3] in speaking about the general treatment of epilepsies, mentions that about 20 per cent. of his cases showed the presence of albumin and casts following attack.

It is worth while to review here the physiologic conception as to how albuminous substances find their way into the urine in disease and in health so that we may be able to justify some conclusions drawn from the study in this paper. Sir William Osler [4] states substantially that under normal conditions of kidney function the glomerular epi-

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1. Simon, C. E.: A Manual of Clinical Diagnosis, Ed. 9, Philadelphia, Lea & Febiger, 1918, p. 383.
2. Nothnagel, cited by Simon, Footnote 1.
3. Munson, J. F., in White, W. A., and Jelliffe, S. E.: Modern Treatment of Nervous and Mental Diseases **2**:266, 1913.
4. Osler, William: Principles and Practice of Medicine, Ed. 8, New York, D. Appleton & Co., 1914, p. 684.

thelial cells allow the passage of only water and salt from the circulating blood, and will not permit albuminous substances to filter through. Injury to these cells by disease results in free passage of albumin and other protein substances. According to all observers, in physiologic albuminuria, the epithelium is not impaired permanently and the occurrence of albuminous matter in the urine is, therefore, attributed to muscular contraction as, for instance, in the course of violent exercise during athletic engagements or possibly during convulsive attacks in certain diseases. Whether it be called physiologic, or as recent writers tend to call it, constitutional albuminuria, all agree that such a condition is probably outside the pathologic domain. Should the albuminuria following convulsive seizures in epilepsy be considered as physiologic?

This paper presents a study of a number of epileptics in our hospital where tests were made under favorable conditions. The study makes no claim to anything new but attempts to clear up a point of disagreement in that direction.

By request of the laboratory a specimen of urine was received from every case of epilepsy, following as soon as the patient regained consciousness after an attack. The specimens were examined as soon as practicable after reaching the laboratory. The presence of albumin was determined by the heat and acetic acid test and the microscopic work was done with a centrifugalized portion, using the flat mirror of the microscope. It was sought to determine the following points:

1. The general frequency of albumin with casts and other microscopic findings in the number of cases under observation. As already intimated, the frequency of positive urinary postseizure findings has been underestimated by some workers, and Simon concludes that albuminuria following seizures in epilepsy is the exception rather than the rule.

2. The length of time subsequent to an epileptic convulsive seizure that albuminuria with casts can be demonstrated. Does only the first urine after an attack contain albumin, or does the albuminuria persist for a longer period? Stated differently, does the period of unconsciousness hold any relation to the urinary findings?

3. The relation of accidental albuminuria, as evidenced by the presence of semen, to albuminuria due to the convulsive seizures per se. As noted above, semen as a contaminating agent in the causation of albuminuria following convulsive attacks of epileptics has probably been given undeserved prominence as an accidental agent. While it is true that in the presence of semen the albuminuria is quantitatively increased, the presence of albumin in the absence of semen is comparatively greater. From the number of examinations recorded in

Table 1, the author is inclined to believe that seminal fluid, as an albuminurious factor in epilepsy following seizures, plays a rôle of no great significance. The following statement by such a reputable observer as Simon is of particular interest in this connection, as it conveys the impression that semen is the only agent causing albuminuria in postepileptic seizures. "The seizures in those cases were comparatively slight and unfortunately an examination for semen was not made in those urines in which traces of albumin were demonstrated." Admitting that there may be present a small quantity of semen in the urine of every epileptic individual subsequent to seizures, we cannot always attribute the albuminuria to this cause, as the quantity of urine voided will readily change the albuminuria index: the greater the quantity of urine, the amount of semen remaining constant, the less the chances for demonstrating the albuminuria with our present qualitative methods. Furthermore, the presence of granular casts along with albuminuria is prima facie evidence that it is not of accidental origin through admixture of semen.

4. The possibility of differentiating true epileptic seizures from hysteria or epileptiform convulsions. Simple and unadorned as such a laboratory procedure might prove to be in such diseases as epilepsy and hysteria requiring psychologic study, this point was nevertheless kept in mind as the examination of the material proceeded.

From Table 1 it will be seen that two cases (P. B. and D. M.), whose urine was examined ten and nine times, respectively, corresponding to the same number of attacks, have shown no albumin and no casts to any degree; they were diagnosed clinically as hysteria. This number of cases is entirely too small for differential diagnostic study. Observation of a large number of cases over a long period would seem essential. Perhaps, taking the view of the general frequency of albuminuria in the number of cases studied as physiologic and apparently due to muscular contraction effected during the seizures, we may probably account for the absence of urinary findings in the hysteria cases, as the hysteric individual often does not present the typical convulsion of the genuine epileptic; the hysteric frequently simulates the epileptic in his seizure manifestations, especially when brought to live in contact with the epileptic. There would, moreover, seem to be no physiologic cause for albuminuria in hysteria as the hysteric is perhaps physically not concerned in his apparently epileptiform spells. This consideration would suggest an affirmative answer to the question brought forth in this paper as to whether the albuminuria with casts in postepileptic seizures is physiologic. The subject of physiologic albuminuria in general would thus find a supporting point. The summarized table also shows that forty cases, or 66 per cent., have shown albumin with casts after every attack; 193 specimens were examined

TABLE 1.—SHOWING THE FREQUENCY OF ATTACKS, DURATION AND NUMBER OF SEIZURES AND URINARY FINDINGS

Case	Average Time After Seizures, Hours	Approximate Duration of Seizures, Minutes	Number of Seizures During Six Months' Residence			Albumin	Casts	No. Times Examined	No. Times Negative	Semen
			Diurnal	Nocturnal	Total					
E. Z.	9	10	1	7	8	+	+	5	2	—
J. E.	3½	10	10	7	17	+	+	3	0	—
S. G.*	12-15	15	7	10	17	+	+	4	0	—
V. McC.	11-19	15	5	3	8	+	+	3	1	—
F. J.	21	15	1	3	4	+	+	1	0	+
W. S.	12	120	3	—	3	—	—	1	1	—
W. R.	2-6	15	37	26	63	+	+	7	0	+
J. D.	11	15	2	1	3	+	+	1	0	—
E. W.	18	10	1	4	5	+	+	2	1	—
F. S.	10-18	10	3	2	5	+	+	2	1	—
J. O'R.	6-7	5	6	15	21	+	+	6	0	—
E. O'C.	15	5	1	0	1	+	+	1	0	+
R. W.	9	10	1	1	2	+	+	2	0	—
R. B.	9	15	2	3	5	+	—	2	0	—
A. P.	10	10	4	2	6	+	+	1	0	—
C. H.	5-12	10	1	7	8	+	+	3	0	—
L. A.	6	10	5	3	8	+	+	3	0	—
G. W.	9-12	50	8	7	15	+	+	4	0	—
E. C.	8	15	26	14	40	+	—	3	0	—
C. L.	4-9	15	5	4	9	+	+	3	0	—
D. S.	6	10	2	4	6	+	—	2	0	+
J. L.	8	15	1	2	3	—	—	1	1	—
F. F.	11	15	1	4	5	+	+	2	0	—
A. M.	8	2	49	21	70	+	—	2	0	—
J. S.	12	120	21	9	30	+	+	1	0	—
F. J.	12	7	0	1	1	+	+	1	0	—
R. W.	?	20	0	6	6	+	+	1	0	—
H. G.	13	15	0	2	2	+	+	1	0	—
H. B.	14	30	4	4	8	—	+	1	0	—
B. D.	?	60	17	17	34	+	+	2	0	—
W. L.	1-10	15	29	5	34	+	+	10	0	—
J. R.	3-9	10	1	4	5	+	+	4	0	+
C. D.	9	20	1	3	4	+	+	1	0	+
J. B.	9-10	20	15	39	54	+	+	3	0	+
B. H.	9	20	3	0	3	—	—	3	3	—
O. B.	6	15	13	4	17	—	+	2	1	—
D. P.	21-24	15	4	1	5	—	—	2	2	—
W. E.	4-24	10	18	8	26	+	+	4	2	—
P. W.	4-15	15	17	11	28	+	+	6	2	—
A. C.	15	15	11	15	26	+	+	3	1	—
E. W.	7	10	1	3	4	+	+	2	0	—
J. P.	6-9	15	1	7	8	+	+	3	1	—
H. C.	6-12	15	5	10	15	+	+	4	1	—
C. W.*	8-9	10	15	30	45	++	++	10	0	—
L. S.	3-9	10	2	8	10	++	++	5	1	—
E. M.	3-6	15	3	25	28	++	+	6	0	—
G. L.	4	10	4	4	8	+	+	2	0	—
T. E.	4-9	15	1	11	12	++	+	6	0	—
D. M.	12-18	8	9	15	24	—	—	9	9	—
W. T.	4-6	15	7	25	32	++	+	8	0	+
W. C.	9	10	17	13	30	+	+	1	0	—
J. F.	6-13	20	25	6	31	+	—	2	1	+
W. B.	5	20	1	4	5	+	+	2	0	+
E. J.	12-16	10	15	9	24	+	+	3	2	—
W. H.	10-12	12	2	61	63	+	+	9	0	+
R. H.	8-15	15	8	10	18	+	+	7	5	—
L. A.	12	10	55	5	60	+	+	6	0	—
D. A.	22	10	7	—	7	+	+	—	0	—
M. W.	14	3	1	1	2	+	+	1	0	—
H. H.	3-10	15	1	3	4	+	+	3	0	—
S. P.	3-5	10	1	2	3	+	+	3	0	+
P. B.	6-9	5	14	31	45	—	—	10	10	—

* Positive Wassermann reaction. + = Beyond a trace. ++ = Appreciable trace.

with an average of 3.1 per patient. In thirteen cases the urine was positive after some attacks and negative after others in the same individual. While the regularly positive cases include several whose urine was examined after one or two attacks and perhaps would have become irregular as far as the urinary findings are concerned, it is believed that the irregular cases would have been considerably reduced if in many instances the first urine voided after an attack had reached the laboratory. A patient who is seized in the early evening hours is likely to have voided once before the next morning's specimen is taken. Frequent changes in attendant personnel on the wards would tend toward the same error. I have not been able to find any relation between frequency of seizures and regularity of positive findings in the urine subsequent to the seizures. Table 2 shows nine cases whose frequency of attacks during a period of six months' residence in the hospital ranged numerically from 21 to 70. At no time after seizures was the urine free from albumin and casts. Several of these cases have tended to persist in showing abnormal urine for from twenty-four to forty-eight hours subsequent to the attack. It seems reasonable to assume that very frequent seizures would tend to produce an unfavorable renal condition. This fact was emphasized by Munson when speaking of specific therapy for those giving indications for treatment.

TABLE 2.—Selected Cases from Table 1. Frequency of Seizures During Six Months in Hospital, and Urinary Findings

Case	Number of Attacks	Number Urine Positive	Number Urine Negative	Persistence of Urinary Abnormalities. Hours
F. J.	1	1	0	Negative
J. O'R.	21	6	0	Negative
E. M.	28	6	0	24
W. T.	32	8	0	24
W. L.	34	10	0	24
C. W.	45	10	0	48
J. B.	54	3	0	24
W. H.	63	9	0	48
W. R.	63	7	0	24

TABLE 3.—Summary of Table 1

Total Number Cases	No. Cases Regularly Positive	No. Cases Regularly Negative	Number Irregular Cases	Total No. Specimens Examined	Average per Patient	No. Cases Semen Present
60	40 (66%)	4 (6.6%)	13 (21.6%)	193	3.1	12 (22.6%)

A glance at Table 3 will also show that the incidence of semen with albuminuria as a probable factor of contamination was found to be 22.6 per cent. in a series of sixty cases. While these cases have shown a slight increase in the quantity of albumin over those whose urine did not contain that element, casts were simultaneously demonstrated. Other quite constant findings were those of amorphous urates and phosphates. Since the material was examined during the winter months, these findings would be of no significance. I have not been able to demonstrate glycosuria in a number of the specimens obtained after attacks, though some workers have reported sugar to be present in the urine in epileptics following seizures.

CONCLUSIONS

1. In a series of sixty cases of epilepsy, two-thirds, or 66 per cent., have shown albuminuria with granular casts after every seizure. Two cases of this series who were diagnosed clinically as hysteria, gave no evidence of either albumin or casts in the urine after repeated examinations following epileptiform seizures.

2. Albuminuria with casts persist for from twenty-four to forty-eight hours subsequent to attacks in some genuine epileptics.

3. The frequency of seizures, while apparently holding no relation to positive urinary findings, tends in general toward regularity of urinary abnormalities after attacks.

4. The duration of seizures seems to bear no relation to subsequent albuminuria.

5. Seminal fluid as a contaminating factor giving rise to albuminuria after epileptic seizures does not play a very great rôle.

6. An investigation into the frequency of nephritis in epilepsy is suggested.

My thanks are due to Mrs. Mary E. Aucoin, chief nurse of the hospital, for her cooperation and kind assistance.

CORRECTION

In the article by Dr. Charles Frazier, which appeared in the August issue of the ARCHIVES OF NEUROLOGY AND PSYCHIATRY, the tracts in the illustration of the cord, on page 139, were incorrectly outlined. This illustration was inadvertently sent with the manuscript. The correct illustration appears in the reprint, a copy of which may be secured by sending a stamped, addressed envelope to the author.

The author also calls attention to an error in his manuscript: "latterly I have chosen the sixth cervical segment." This appears in the nineteenth line from the top of page 140. It should read: "latterly I have chosen the sixth thoracic segment."

Editorial

COMPENSATION AND "CONDUCT DISORDERS"

Among the many excellent points brought out in Dr. Dana's article on "Wounds of the Head and Compensation Laws," the one that refers to final compensation seems to be of especial importance. There should be no objection to final compensation by the state for such injuries as can be definitely measured or determined; for the loss of an eye or a limb, a prompt final settlement seems to be the most suitable compensation.

It is not of these cases, however, that Dr. Dana writes. On the contrary, the symptoms complained of by the majority of the survivors of head injuries are rarely of a character to be evaluated accurately by the physician, except in so far as he believes the statements of the patient or of his witnesses. They belong to the order called functional, or to "conduct disorders" as Dana prefers to call them, and, after a reasonable time for rest has elapsed, the sovereign remedy for them is *morale*. A neurotic conduct disorder is maintained with the idea, conscious or subconscious, that some benefit will be derived from it. When that idea is removed, the neurotic patient makes a much better adjustment. For the good of the patient and the benefit of the commonwealth, in appropriate cases, it is the duty of the state, when compensation is sought, to combat that idea from the outset. The state would do so if competent neurologists were its advisors; but they rarely are. Neurologists must take a more active interest in legislative affairs before legislators will be willing to allow them to decide that certain fairly definite symptom groups are exceptions to the general rule, and that the patients are injured by compensation. The legislators might be convinced individually, but it is more difficult to convince them as a group. And yet what inestimable benefit France did itself and its soldiers by deciding that purely functional cases were not eligible for pension!

P. B.

News and Comment

FIRST ANNUAL MEETING OF THE ASSOCIATION FOR RESEARCH IN NERVOUS AND MENTAL DISEASES

The first annual meeting of the Association for Research in Nervous and Mental Diseases will be held during the Christmas week of 1920 in New York City. The subject for discussion will be "The Acute Nonsuppurative Infections of the Nervous System." The exact date and place of the meeting will be announced in the December number of the ARCHIVES and full particulars, including the program, will be published in that issue.

The secretaries of all interested neurologic or psychiatric societies may obtain the necessary blank forms for membership application from the secretary-treasurer, Dr. Foster Kennedy, 20 West Fiftieth Street, New York City. All members in good standing in any neurologic or psychiatric society are eligible to membership on payment of the annual dues of $5. This fee entitles the member to a copy of the transactions, which will be published by the Commission of the Association after the meeting.

HARVARD PROFESSORSHIP IN PSYCHIATRY

Dr. Charles MacFie Campbell, assistant director of the Henry Phipps Psychiatric Clinic, Johns Hopkins Hospital, has severed his connection with the clinic in order to become professor of psychiatry at Harvard Medical School and director of the Boston Psychopathic Hospital. Dr. Campbell will assume his new duties, October 1.

Abstracts from Current Literature

STUDIES ON ARTHRITIS IN THE ARMY BASED ON FOUR HUNDRED CASES. I. PREAMBLE AND STATISTICAL ANALYSIS. RALPH PEMBERTON, with the assistance of JOHN W. ROBERTSON, Arch. Int. Med. **25**:231 (March) 1920.

A decision of the surgeon-general permitted a study of chronic arthritis on a scale larger than has been possible in this country since the Civil War.

Statistical Analysis of Four Hundred Cases of Arthritis Admitted to the U. S. Army General Hospital No. 9, Lakewood, N. J.—It was found that 64 per cent. of the patients had arthritis only; 28 per cent. had a combination of arthritis and myositis; 5.5 per cent had myositis only; 1.75 per cent. had nerve involvement (neuritis) only, and 0.75 per cent. were listed as doubtful. The average age was 28.26 years, while that of the worst cases showing the least improvement was 29.38 years.

TABLE 1.—PRECIPITATING FACTORS IN ORDER OF FREQUENCY

	No. Cases	Percentage
Exposure	232	58.0
Dysentery	33	8.25
Injury	30	7.5
"Flu"	28	7.0
"Gas"	23	5.75
Drilling and hiking	15	3.75
Tonsillitis	13	3.25
Pneumonia	6	1.5
Neisserian infection	4	1.0

One hundred and forty-three patients (33.75 per cent.) had had attacks of "rheumatism" prior to army service. Only eight of 113 nonarthritis patients on the surgical and orthopedic wards, questioned at random, had had such attacks.

TABLE 2.—DISTRIBUTION OF INVOLVEMENT

	Percentage
Knee	62.0
Ankle	35.25
Hip	33.73
Shoulder	31.75
Legs (muscle)	22.25
Hand	14.50
Spine	13.50
Foot	12.0
Wrist	11.75
Elbow	9.75
Back (muscle)	8.25
Arm	7.50
Thigh	7.0
Ileosacral joint	4.0
Heel	1.75
Sciatic nerve	1.0
Jaw	0.75
Lumbosacral joint	0.50
Facial nerve	0.25

It was noted that 57 per cent. showed a combination of two or more of the above distributions.

Relative to Surgical Foci.—One hundred and seven persons (26.75 per cent.) were taken sick in the apparent absence of demonstrable surgical foci; 73.25 per cent. showed demonstrable surgical foci. Of the latter number, 71 per cent. showed foci in the tonsils and 45.73 per cent. of those showing a demonstrable surgical focus were positive for a dental focus. Seventeen per cent. of those showing a focus were positive for a genito-urinary focus. Of the 293 persons showing a focus in any of the above three distributions, 26.62 per cent. showed a combination of both dental and tonsillar foci, and 13 per cent. some combination of foci other than dental and tonsillar.

TABLE 3.—REGARDING PROGRESS

Percentage	Remarks
23.0	Recovered in the apparent absence of a demonstrable focus
46.0	Recovered in the presence of a demonstrable focus
8.50	Recovered after the removal of foci
7.75	Improved after the removal of foci
7.0	Unimproved after the removal of foci
77.75	Recovered "by whatever means"
18.50	Improved "by whatever means"
4.0	Unimproved "by whatever means"

It seems to be quite clear that after making all allowances for error, exposure to cold and wet is a factor of major importance in producing rheumatoid and arthritic disabilities among soldiers in service under present conditions of warfare. After considering all factors, it would seem that mechanical agencies, such as trauma in marching, played a rôle not conspicuously greater than they do in civil life.

The fact that 184 patients (46 per cent.) recovered in the presence of demonstrable foci appears somewhat at variance with the relation focal infections are known frequently to bear to the disease but should not be interpreted as justifying neglect of this established rôle. It is probable that a distinct contrast to the conditions influencing chronic arthritis in civil life is here evidenced, and that the age of the subjects and the recuperative powers of youth must be given large significance. It seems safe to conclude that genito-urinary infections played a small rôle in the causation of chronic arthritis in the army.

II. OBSERVATIONS ON THE BASAL METABOLISM. Ralph Pemberton and Edna H. Tompkins, Arch. Int. Med. **25**:241 (March) 1920.

Some evidence has indicated that a variety of agents supposed to hasten body metabolism, such as thyroid extract, radium and the roentgen ray, may exercise definitely beneficial effects on chronic arthritis and that certain patients do much better on a lowered food intake, particularly of carbohydrate. These and other considerations have suggested that there might be in chronic arthritis a lowering or retardation of body metabolism which would express itself in summation figures. The basal metabolism was, therefore, studied in a series of twenty-nine cases. Of these, 80 per cent. showed a metabolism within normal limits; in 20 per cent. it was slightly below normal.

III. STUDIES ON THE NITROGEN, UREA, CARBON DIOXID COMBINING POWER, CALCIUM, TOTAL FAT AND CHOLESTEROL OF THE FASTING BLOOD, RENAL FUNCTION, BLOOD SUGAR AND SUGAR TOLERANCE. Ralph Pemberton and Goodwin L. Foster, Arch. Int. Med. **25**:243 (March) 1920.

Nitrogen and Urea of the Blood.—In previous communications evidence was advanced to show that there is no disturbance of the fasting level of the blood urea or nonprotein nitrogen in cases of chronic arthritis. Determinations of blood nitrogen and urea, conducted on seventeen patients of this series, show that all cases, including those of the most severe type, fall well within normal limits. These conclusions were confirmed by Lieut. Thomas E. Buckman on forty other patients.

Carbon Dioxid Combining Power of the Blood.—Studies were conducted on seventeen cases in respect to the carbon dioxid combining power of the blood as an index of acidosis. In every case the figures fell well within normal limits, the lowest being 53 and the highest 67.

The Calcium of the Circulating Blood.—In view of the known absorption and deposition of lime salts in chronic arthritis affecting osseous tissues, observations were made on the calcium in the fasting blood in various types. Ten cases were studied, and the results were with surprising uniformity within normal limits. The cases studied were mostly of great severity, although three patients of the series represented milder types.

The Total Fat and Cholesterol of the Fasting Blood.—Of late a considerable field of investigation has been opened up in connection with the blood fats and chlosterol and their relation to metabolism, more particularly since the introduction of Bloor's simpler analytic methods. Data in a variety of pathologic conditions tend to place certain phases of the metabolism of fat on a basis analogous to that obtaining with the nitrogen and urea of the blood. Studies were, therefore, conducted on the total fats and cholesterol of the fasting blood, using the method of Bloor. Determinations were made on fourteen subjects, but gave results which fall within the rather wide range attributed to normal values. All degrees of severity of the disease were represented.

The Renal Function.—Until the rôle played by focal infections was appreciated, it was the custom, and still is in many quarters, to attach chief importance in the therapesis of arthritis to stimulation of eliminative functions, particularly those of the skin. While such measures are useful, they rarely remove the cause of the disease, and the results attributed to them are sometimes referable to other incidental or intended factors.

Of the several tests of kidney function, the method depending on the test meal of Hedinger and Schlayer, modified by Mosenthal, probably gives the earliest indications of diminished kidney efficiency. It has not been applied hitherto to the study of arthritis in a large way. This test depends on the eliminative response of the kidney to approximately constant amounts of water, salt and nitrogen ingested in a generous mixed dietary of about 2,100 calories. We, therefore, adopted this test as a further means of studying the renal function in a considerable series of arthritics and normal persons.

The results of this test indicate that individual cases of arthritis occasionally show low outputs of water for the twenty-four hour period, and sometimes retention of salt and nitrogen. The mean water output of twenty-nine arthritic patients shows a normal total volume of 1,200 c.c.; the mean salt output an approximately normal amount of 11.36 gm., and the mean nitrogen output an approximately normal amount of 11.1 gm. The rather wide range allowed to normality makes it difficult to attach importance to small departures from it. Comparison of these figures with those from normal persons shows on the part of the arthritic patients a mean water output about the same as that of normal persons, a mean nitrogen output slightly smaller, and a rather lower mean salt output.

Observations were also conducted on phenolsulphonephthalein elimination. The results were all normal. Hanzlik, in studying the salicylates, has found phenolsulphonephthalein values ranging from 55 to 75 per cent., and has concluded that renal functional efficiency is quite as good in the rheumatic as in the normal individual.

The Blood Sugar.—It has been shown by one of us that in a considerable percentage of cases of chronic arthritis there is a definite relation between the intake of food and the incidence or perpetuation of symptoms of the disease. This relation is best illustrated by the fact that in appropriate cases the institution of a reduced diet may be followed by marked benefit. Of the three foodstuffs, the evidence at hand indicated that carbohydrate is most concerned in this connection. It became pertinent, therefore, to determine whether there is difficulty in the metabolism of carbohydrate, and to this end studies were carried out in sixty cases of arthritis on the fasting level of the blood sugar and on the response of these cases to the so-called glucose tolerance test. The glucose tolerance test was conducted according to the method of Hamman and Hirschmann. In the present series the renal threshold for glycosuria fell between 0.17 per cent. and 0.18 per cent., which agrees with the values found by others for the normal kidney, and is something of an argument for the normality of renal function in arthritis, as the threshold may be much higher in true nephritis.

Glucose tolerance tests were first conducted on a series of chronic arthritic patients, beginning with the most severe types, and as it became apparent that there was in most of them a marked difference in their response, our observations were extended to a large number of cases of all types. In general, it can be said that the height of the sugar curve, that is to say, the degree to which the sugar tolerance is lowered, is largely proportional to the severity of the active process *per se.* Severity of the process apparently plays as big a rôle as does the extent of the process in affecting the tolerance. Some of the curves fall within or only slightly above normal values. Ordinarily these exceptions occur in patients who have passed the zenith of their trouble and who show a tendency to convalesce, or they occur in cases in which the distribution and severity are limited. But the parallelism is not exact. It should be borne in mind that the glucose tolerance test is merely a gross way of ascertaining deviation of a function which is probably complex and dependent on factors not understood, and that it is not necessarily adjusted to reflect lowered tolerance in all cases.

A group of twenty convalescents was then studied. These had had a severe and generally long continued arthritis and at the time were free from symptoms. It was found that this group gave curves of a lower average than the severe or moderate cases; that is, 74 per cent. of the group of convalescents gave a normal tolerance.

Investigating the relation of this disturbance to some of the other factors known to influence chronic arthritis, we conducted a series of determinations on the tolerance during the ill health and convalescence of the same person, following various procedures. It was found that there was an abrupt change in the sugar tolerance in patients who improved after removal of apparently causative foci of infection. Thus there was a striking contrast after twenty-nine days in the sugar tolerance of a soldier, the subject of intense arthritis of long standing in the left hip and foot, who made an abrupt and rapid convalescence following tonsillectomy. A curve showing normal tolerance was obtained fourteen days after the tonsillectomy.

The group of men convalescing in the presence of demonstrable surgical foci of infection suggests that the disturbed tolerance may return to normal as the patient improves, even without the removal of the focal infection. At least five persons giving normal tolerance curves were the subjects of demonstrable surgical foci. One case (Case 22) illustrates a distinctly lowered sugar tolerance (high curve) observed in a patient who later made striking improvement, because of a sharp dietary, in the presence of a surgical focus. The curve next to the highest was that obtained when this soldier was at the height of his trouble, which consisted in almost complete ankylosis of both hips, knees and spine, together with great limitation of function in the left shoulder. The third and fourth highest curves were obtained when the shoulder had made an emphatic therapeutic advance following diet. The lowest curve represented the return of the sugar tolerance to practically normal limits, following the removal of necrotic tonsils, after he was well on in convalescence and able to walk with crutches.

Therefore it seems clear that as patients with arthritis improve, irrespective of the therapeutic measure to which this is due, the lowered sugar tolerance tends strongly to a return to normal. It seems to do so most rapidly, however, after surgical removal of the causative infection. Our experience suggests that the sugar tolerance test may sometimes be helpful to indicate whether all foci have been removed; for example, a case of active arthritis of moderate chronicity may fail to improve after the removal of dental foci. If the tonsils in such a case are apparently normal, as often happens, the maintenance of a definitely lowered sugar tolerance would suggest that foci are still operative and presumably in these tissues. It must be borne in mind that long standing cases from which all demonstrable foci have been removed may retain their lowered tolerance.

Among three supposedly healthy persons, the subject of marked tonsillar pathology without symptoms, one curve was slightly elevated above the normal, and the others fell within normal limits. One of these patients showed such gross pathology that his tonsils were removed a day or two later as a prophylactic measure. It is important, therefore, to note that, by and large, the disturbance of the sugar tolerance, as here considered, due to focal infection, apparently accompanies the failure of the organism to maintain successfully its wall of defense and is apparently restored to normal when this defense returns. In this light a lowered tolerance, following a focus, becomes an intermediary, or, at least, a concomitant step in the pathology of arthritis and possibly other conditions as well.

Lowered sugar tolerance is specific for no disease and has been noted in a variety of conditions. It is important that the results of sugar tolerance tests in as many different diseases as possible be recorded, and there were, therefore, included in the present study determinations of the sugar tolerance in twelve cases of miscellaneous disease at large, as they presented at the hospital. These showed that patients with severe arthritis gave the highest curves, the miscellaneous pathologic cases the next highest and patients with moderate arthritis the next; the mild and convalescent arthritic patients followed with curves of about the same height; the curves in the normal cases being the lowest. It is probable that selection could be made of certain types of miscellaneous pathologic cases which would determine such a composite curve at either a higher or a lower level, according to the groups chosen.

It has generally been believed that, with the exception of disease of the pancreas, disturbances of the thyroid are the most common cause of altered carbohydrate tolerance. However, even in hypothyroidism sugar tolerance shows perplexing combinations, although in a general way low in hyperthyroidism and high in hypothyroidism and individual patients display wide latitude in their responses. The same conditions obtain in disturbances of the hypophysis. It has also long been known that in nephritic patients the blood sugar often is unusually high. However, as Hamman and Hirschmann remark: "It is not always high; sometimes it is at the normal level, and why some cases have hyperglycemia and others have not has never been satisfactorily explained."

It would seem in the light of the evidence herewith adduced that lowering of the sugar tolerance, as exemplified by the glucose tolerance test, rests, in part at least, on somewhat more fundamental pathologic processes than has hitherto been suspected. It seems to stand in some relation to inflammatory processes and it appears from the data adduced herewith that the presence of an inflammatory process may induce a lowered sugar tolerance; that is, a high curve. Other curves definitely prove that marked reduction of sugar tolerance may abruptly return to normal after the removal of a focus of infection. It is possible that some departures from normal observed in studying nephritis, thyroid and hypophysial disorders, etc., are referable to such agencies as focal infection. This applies, of course, to the miscellaneous diseases the authors have studied and suggests the inexpediency of extending such observations unless carefully controlled. Thus a high curve afforded by a case of plumbism may have been due to a focus that we did not suspect.

A lowered sugar tolerance, as measured by the glucose tolerance test, seems to be a more common occurrence than has been appreciated. In view of the demonstrated relation of focal infection to sugar tolerance; in view of the frequency of foci in arthritis and at large; in view of the frequency of arthritis as a disease and the frequency with which it is accompanied by a lowered sugar tolerance, it seems necessary to modify the view which ascribes to disturbance of thyroid function, next to diseases of the pancreas, the most common cause of altered carbohydrate tolerance. It is clear, however, that most arthritic patients are unable to remove from the circulating blood, as does a normal person, the carbohydrate that reaches it from the gastro-intestinal tract.

IV. STUDIES IN THE RELATION OF CREATIN METABOLISM TO ARTHRITIS. Ralph Pemberton and Thomas E. Buckman, Arch. Int. Med. **25**:335 (April) 1920.

The researches of the past fifteen years on the chemistry and biochemistry of creatin and creatinin have abundantly established the high importance of these substances in the animal economy. More or less characteristic glucose intolerance has been demonstrated by Pemberton and Foster in cases of arthritis. As creatinuria has been demonstrated in hyperthyroidism (after high protein feeding) and in other conditions accompanied by a disturbance in the body's capacity to utilize carbohydrate, it was deemed worth while to investigate a series of arthritic patients with a view to determining the presence of abnormalities in the metabolism of creatin as revealed by blood and urine analysis.

Summary.—1. In determinations of the creatin, creatinin and nonprotein nitrogen of the blood and urine of forty cases of arthritis and in nine normal patients used as controls, about one-half of the cases of arthritis showed an abnormally high value for blood creatin.

2. A certain number of patients showed a decline in blood creatin simultaneous with clinical improvement.

3. Only three of the cases showed creatinuria.

4. Only two cases showed abnormal elevation of nonprotein nitrogen of the blood.

V. ROENTGEN-RAY EVIDENCES, CLINICAL CONSIDERATIONS, TREATMENT, SUMMARY, CONCLUSIONS AND CLINICAL ABSTRACTS OF CASES STUDIED. RALPH PEMBERTON, Arch. Int. Med. **25**:351 (April) 1920.

Roentgen-Ray Evidences.—Roentgen-ray studies were made in all cases of the 400 in which there was any suspicion that there might be additional evidence discoverable by these means. One hundred and seventy-nine series of plates were made, ranging from one to twenty plates in any given series, and the percentage of positive findings justified omitting recourse to the roentgen rays in the absence of such suspicion. Making allowances for cases which may have been overlooked, a relatively small number of the entire series, less than 15 per cent., showed evidences of bony change when examined by the roentgen ray. It is clear that arthritis and its accompanying phenomena may exist for many months, producing gross superficial changes, without evidence that the roentgen ray can detect, except in the increased shadows of the soft parts.

Clinical Considerations.—Cases occurring under military conditions differ from those in civil life chiefly in respect to the age, the immediately exciting factors and the greater tendency to improve. Nearly all types encountered in civil life were represented, although the incidence was different. The age of the patient and the relatively limited chronicity of the disease precluded the occurrence of gross lesions of the degree and frequency encountered in the later decades of life. Although chronic arthritis in the young is not rare, and some of the most refractory cases occur at this age, its frequency is vastly increased by war conditions.

On account of the lesser frequency of structural bony change, it was sometimes difficult to decide as to the existence or degree of disability. Cases involving the spine and sacro-iliac joints presented the greatest difficulty in diagnosis. The minor neuroses secondary to illness and war conditions often complicated the picture. A certain number of cases showed acute tenderness to pressure at and below the costal borders posteriorly, suggesting, but not simulating, lumbago.

A small number of cases called to mind the picture presented by certain supposedly neurotic conditions, unaccompanied by organic change, in which the individual bends forward as though the subject of advanced vertebral disease. Under the heading "camptocormie," "spondylose antalgique" and other titles, the French have directed considerable attention to this condition, and the diagnosis between it and spondylitis deformans is acknowledgedly difficult. We concluded that in the presence of disabilities referred to the back as a whole, it was unsafe to deduce without prolonged, exhaustive observation that organic disease was not present.

The exciting factors other than exposure need a little comment. It is somewhat surprising that dysentery should occupy the second place. The arthritic attack came on at all stages of the apparently causative disease, even occasionally nearly or quite coincident with convalescence, when the conditions of life were not adequate to explain it.

It is, of course, entirely possible, theoretically, that when exposure acted as the exciting agent in producing arthritis, it did so through the intermediation of a focus of infection by lowering the general resistance to such infection or by favoring the growth of such a focus. We believe that the question is not yet settled and that we must admit the possibility that exposure per se is capable of inducing "rheumatic" disability.

It is difficult or perhaps impossible at present to draw a line sharply between chronic arthritis which includes occasional acute febrile manifestations and acute inflammatory rheumatism per se, and several cases of this series appeared to merge from one condition into the other. In acute inflammatory rheumatism circulating bacteria may cause valvulitis, emboli and sepsis; and true sustained inflammatory rheumatism with valvulitis is probably due to bacterial infection only. In both conditions the arthritic phenomena probably result from some common intermediary step, such, for example, as that productive of a lowered carbohydrate tolerance secondary to focal infection or other agencies. The chronic and acute types differ in degree rather than in kind.

Treatment.—A large number of patients were recovering on admission or had recovered following nothing more fundamental than rest and external measures, notwithstanding severe and protracted invalidism. These facts, together with our own experiences, forced on us consideration of the results obtained by the expectant plan of treatment when based on such measures as baking, massage, hydrotherapy, electricity and the like. They were much more encouraging than those encountered in civil life. The kinds of treatment that received greatest emphasis were, first, removal of foci of infection, and second, local and external measures. Other forms of therapy figured less conspicuously. Thirty-four patients, or 8.5 per cent., recovered after the removal of foci. Thirty-one patients, or 7.75 per cent., improved after the removal of foci, but the end-result may have been favorable in certain unimproved cases that disappeared early from our observation.

This series has clearly indicated an impressive independence of focal infections as compared with older subjects. But it must be granted that the conservative step in a refractory case is removal of focal infection, and apparent normality of the tonsils is not unfailing evidence that they are not acting as focal agents. For practical purposes it is wiser to regard the relative independence of focal infections shown by these young subjects as an academic consideration, and to regard possible foci of infection as causative agents until proved otherwise.

The removal of dental foci also played an important rôle, but improvement was rather more striking after tonsillectomy. Genito-urinary disease afforded only a small percentage of the foci.

Nonspecific Protein Injections.—This form of therapy, as applied to arthritis, achieves its best results in the acute forms. The patients in the present series, to whom this treatment was given, belonged for the most part in the chronic category. The army typhoid vaccine was used in an initial dose of twenty-five million, the second dose, when given, being fifty million and the third, seventy-five million. Nineteen patients received nonspecific protein injections in a vein of the arm. Of these, seven were definitely improved, ten were unimproved and in two the results were uncertain. Although nonspecific protein in the form of typhoid vaccine is of some benefit on diffuse groups of arthritic patients, the subcutaneous injections, as practiced routinely in the army camps on the incoming draft, have been without effect to prevent rheumatoid disabilities. Some men had received as many as fifteen injections of the typhoid vaccine prior to the onset of arthritis.

Dietary Considerations.—It has been pointed out in previous contributions that treatment by these dietary measures (sharp curtailment of food intake) should be reserved almost exclusively for cases demonstrably not caused by infectious foci, cases in which the removal of foci is contraindicated, cases not accompanied by under nutrition, anemia, etc.

Case 1.—Robbins, aged 25 years, had suffered from an attack of inflammatory rheumatism nine years previously. After some disability in one shoulder for more than two months, he developed a painful and swollen left ankle. Observations of the food intake for a period of about a week showed ingestion of about 3,750 calories per day, including about 700 calories from candy. Of this, about 12 per cent. came from protein, 29 per cent. from fat and 50 per cent. from carbohydrate. December 3, he was placed on a diet of 2,051 calories, of which about 10 per cent. came from protein, 52 per cent. from fat and 38 per cent. from carbohydrate. Four days later he was much improved. After eleven days the improvement was very marked. After this abrupt change there followed gradual and progressive improvement. His food was then increased to about 2,500 calories, of which about 8 per cent. came from protein, 62 per cent. from fat, and 30 per cent. from carbohydrate. At this point he was apparently in perfect health. The sugar tolerance displayed by this patient on four occasions illustrates that a lowered sugar tolerance may return to normal, coincident with improvement of the individual by restriction of diet.

Local and External Measures.—The subjects of this study showed a surprising response to local and external measures, comprising chiefly baking, massage, hydrotherapy and electricity. These measures all have at least one element in common: the induction of hyperemia. And their field of application is properly in cases which tend to recovery, cases of mild degree and cases in which the basis of convalescence is already laid. They can also be used with propriety in adding to the comfort of more severe refractory types in which, however, their curative effect is slight or absent.

Conclusions.—1. Soldiers developing chronic arthritis have had previous attacks with a frequency about five times greater than have soldiers admitted to hospital for other conditions.

2. Exposure was the exciting factor in 58 per cent. of 400 cases studied. There were apparent foci of infection in 72 per cent., but the present group showed a considerable independence of it. Forty-six per cent. recovered in the presence of demonstrable surgical foci. This is nearly three times the number that improved after the removal of foci. The tonsils were most frequently the site of infection, the teeth were next, the genito-urinary tract came last and clearly played an almost negligible rôle.

3. The sites of most frequent involvement were the knee, the ankle, the hip and the shoulder. All things considered, however, it is not clear that trauma to weight bearing parts played a much greater rôle than it does in civil life in determining the site involved.

4. The basal metabolism was found to be normal in 80 per cent. of twenty-nine cases studied. In 20 per cent. it was slightly below normal limits.

5. The carbon dioxid combining power of the blood; the total fat; the cholesterol and the calcium of the fasting blood were found to fall within accepted normal limits.

6. About one-half of forty cases of arthritis studied showed an abnormally high value for blood creatin. Certain of these showed a decline in blood creatin coincident with clinical improvement.

7. The urea of the fasting blood in seventeen cases fell within normal limits. The nitrogen of the fasting blood in sixty-seven observations in fifty-seven cases fell within normal limits, with two exceptions.

8. Studies of the renal function, in thirty cases of arthritis of widely different types, gave results that fell within the accepted ranges for normal persons. When compared with nine normal persons under similar conditions, there is evident a slight lag in the elimination of water, nitrogen and particularly of salt.

9. A lowered sugar tolerance was found in a large proportion of cases. This lowered tolerance accompanies the great majority of severe cases and is roughly proportional to the activity of the arthritic process per se.

10. The return to normal is apparently independent of the type of therapy employed, but is most abrupt after the removal of causative foci of infection. In certain severe chronic cases from which all demonstrable foci have been removed, a lowered sugar tolerance seems to persist.

11. Apparent foci of infection, unproductive of systemic effects, are not necessarily accompanied by a lowered sugar tolerance. A lowered sugar tolerance from focal infection apparently accompanies the failure of the organism successfully to maintain its wall of defense. In this light a lowered tolerance becomes an intermediary step in the pathology of arthritis and possibly other conditions as well.

12. A lowered sugar tolerance seems to stand in relation to many infectious or inflammatory conditions at large, and to depend on more fundamental pathologic processes than has been appreciated. It is also of more common occurrence than has been recognized.

13. The lowered tolerance observed in some diseases and referred to them may sometimes have been due to focal infection rather than to the diseases under consideration.

14. Critical examinations of recruits for a history of previous attacks of arthritis would reveal cases most likely to develop it. It is reasonable to believe that rejection of this group or at least the worst cases in it would reduce the incidence of arthritis in the army. A more conservative policy would segregate such patients, examine them for foci of infection and remove such foci when found. This would have the added importance of prophylaxis toward the civil community.

15. Local and external therapeutic measures have unexpectedly large application because of the tendency of this group to improve under favorable conditions.

In some cases of this series six months or a year were needlessly lost. Earlier and more critical attention to focal infection as a basis, together with a large coincident use of local measures, would probably afford the routine therapy best adapted to reach the greatest number of cases and should importantly curtail the existing invalidism. Many patients would require more individual attention, however, such as treatment by nonspecific protein injection or a restricted diet.

16. Experience with treatment by a restricted diet corroborates the conclusions previously published. Such therapy finds additional support in the studies on blood sugar, revealing difficulty in the utilization of carbohydrate. It seems clear that success following this measure depends on catering to a weakened function of which the lowered sugar tolerance is one evidence. Treatment along this line has undoubted application in appropriate cases of chronic arthritis.

17. The several measures of value in arthritis should be combined in their application to the present group more frequently than obtains in the treatment of cases in civil life. The tendency to focus on one measure often results in failure where the subsequent coincident use of several measures results in benefit.

Author's Abstract.

AIMS OF BIOCHEMISTRY OF THE NERVOUS SYSTEM. Giacomo Pighini, Neurol. Bull. **2**:395 (Nov.-Dec.) 1919.

Professor Pighini's article is an up-to-date résumé of the work done in the biochemistry of the nervous system, so that the task of condensing an abstract is of somewhat questionable worth; but in view of the interest of the subject an attempt is made to present a few of the salient facts.

After pointing out the limitations of the anatomy and histology of the nervous system and the fallacious information offered by the staining reactions, Pighini speaks of the chemistry of colloids and of the promises which physical chemistry makes in researches on the nervous system. By means of fractional extraction of the lipoids it has been shown that they constitute two thirds of the solids of nervous tissue. The water content is high, 77 per cent. "The formula of percentages of the lipoid groups is constant for every species and for every segment of the examined organ." The cholesterin which exists free and is not combined with fatty acids amounts to 10 per cent. of the total solids. Of the lipoids which have been isolated Pighini mentions leukopolin, cephalin, sahidin, sphyngomyelin, phrenosin, cherosin and cerebroses, or sphyngogalactoses. Of the other solids, the proteins, there are three different globulins, nucleins and amino-acids. The extractives consist of purin derivatives, galactose and inosite.

The gray matter contains a specific nucleoprotein which is absent in the white matter; other substances common to both show only quantitative differences. The gray matter is poorer in lipoids, has less cholesterin and phosphids, much less lipoid sulphur, but more nucleoprotein, neutral sulphur and water. The pons and medulla are rich in nonsaturated phosphids and poor in saturated phosphids. Even the various tracts are said to show differences. As the brain develops the neurons lose some of the water content and gain in lipoids.

These analyses throw light on the chemical reactions of the nervous tissue in health and disease and help to explain the changes in staining reactions. They may also indicate the probable chemical and physical, or biochemical reactions normally going on. Attention is called to the relatively constant (quantitatively) cholesterin, to its rôle as a fixer of the phosphorated lipoids, to its elective fixation of toxic products and to its maintenance of tension of solvents. Cephalin has the property of absorbing oxygen; other sulphurated proteins also participate in the respiratory exchange. Cholesterin, phrenosin or protein fix or neutralize the tetanus toxin; cephalin seems to fix the diphtheria toxin. Lead has a predilection for the ganglion cells of the bulb and pyramids, strychnin for the anterior horns, cocain for the posterior horn, and anaphylactic poisons for the bulb. Elective fixation seems to exist in the case of anesthetics, narcotics, hormones, toxins and animal poisons—all probably on a chemical or chemicophysical basis.

Biochemical studies have been applied in several diseases of the nervous system. Mott and Mann have demonstrated the disappearance of nucleoprotein in amaurotic family idiocy and sought to correlate the fact with the disappearance of the Nissl bodies in the ganglion cells. In dementia praecox Koch and Mann and Pighini found a diminution of neutral sulphur and increase of proteins and inorganic sulphur, also a diminution of lipoid, except cholesterin, and an increase in water. In general paresis there is a decrease of lipoid phosphorus, of the cerebroses and the unsaturated phosphids, while the cholesterin remains constant and the water is increased. Pellagra shows general increase of water, decrease of the lipoids, cerebroses and phosphids, a relative increase of the cholesterin in the cerebellum with slight decrease in the brain and cord, relative preservation of the proteins, a considerable increase of the extractives and a loss of neutral sulphur.

As the degenerative changes in the cells generally seem to follow a definite pattern, chemical analysis will ultimately throw light by means of qualitative and quantitative determinations.

As to chemicophysical behavior, cholesterin does not show the colloidal relation to solvents (chloroform, ether, alcohol, carbon bisulphid, etc.) which cephalin, lecithin and cerebrosids do. Cholesterin raises the superficial tension of organic solvents such as anilin, benzoic acid and naphthalin, while the phosphatids and cerebrosids lower the tension. Various lipoids react differently in the matter of aiding absorption.

Pighini puts forward two hypotheses as to the action of disease products on the nervous tissue. "a. That the pathogenic agent alters definite diastatic agents in the nervous system. b. The pathogenic agent splitting off into aqueous and lipoprotein emulsions becomes attached to certain lipoid components of the protoplasm by chemicophysical means." Narcotics seem to provoke chemical modifications in the brain which show increase of water and diminution of cholesterin.

Pighini outlines the field of investigation and points to the close connection of mineral and hormonic exchange, to radio-active substances in the brain, to ionic action and to the phenomena of polarization and depolarization of cellular elements. He refers to the chemical anatomy or histochemistry of nervous tissue, cellular and plasma fluids, to the superficial energy and tension of the electric potential, to the force and direction of the liquid crystalline molecule, to the dielectric and electromotor force. Through these, he hopes, one will be able to explain the origin of impulses, the manner of their conductivity or interference, and, finally, by means of thermodynamic laws, even psychic energy will be understood.

Wechsler, New York.

A CASE OF DIFFUSE CEREBROSPINAL SCLEROSIS. S. Uyematsu, J. Nerv. & Ment. Dis. **51**:6 (June) 1920.

Dr. Uyematsu reports a case which presented the following symptoms of diffuse sclerosis: The patient had a "stroke" eighteen years before death, followed by eight weeks of aphasia. After this period, there was no speech defect, but she had a slight limp which remained. At 38 she was committed as insane with a diagnosis of manic-depressive psychosis, and showed periods of depression and exhilaration. She had some memory defect and poor insight at the periods of remission. At 48, she was admitted to the Danvers State Hospital showing a positive Wassermann, slight asymmetry of the face and hyperactive reflexes. In a number of years she became demented, gradually weakened, and became indifferent and apathetic. From her forty-sixth year she had occasional fainting attacks followed by unconsciousness. At 49, a tentative diagnosis of paresis was considered, but the spinal fluid was persistently negative. Dementia praecox was also considered as a diagnosis, but never made definitely. She gradually failed physically and mentally until her death.

At necropsy the brain weight was 870 gm. Small cysts were found in the right frontal convolution, in the internal capsule on the right and in the pons. Areas of softening were located in the second frontal convolution, the large part of the centrum semiovale, the internal capsule of the right side and the left lenticular nucleus and internal capsule, and in the pons between the pyramidal tracts and lemniscus bilaterally.

Microscopically, the striking features were alterations in the myelin sheaths and glia cells. In the areas of softening, there was incomplete myelin degeneration and the line of demarcation between the softening and normal tissues was indistinct and irregular, contrary to the usual condition found in diffuse sclerosis. The myelin sheaths seemed to have degenerated hand in hand with the axis cylinders, and in the areas of cyst formation both had entirely disappeared. Pyramidal tracts showed secondary degeneration.

The glia changes were the most important findings. The glia cells were enormously increased throughout the whole central nervous system, their nuclei were unusual in size and shape. In the superficial cortex layer, spider cells, small cells and rod cells were found. In the 3-4 cell layer large numbers of rod cells were present with abnormally elongated nuclei having rounded ends. In the deepest part of the cortex short rod cells and small round nuclei of various forms were numerous. Many rod cells occurred as satellites of ganglion cells and showed no direct relationship to the vessels.

Commenting on the literature concerning rod cells and their origin, the author believes in a gliogenous theory because:

1. They are found together with abnormally increased glia cells.
2. There are all the transitional forms between rod and glia cells.
3. There are many satellite rod cells.
4. There is no proof of relationship with pial vessels.
5. They are found in the cortex where the vessel alterations were not remarkable.

In the peduncle, pons, medulla and cerebellum, the gliosis was most marked, the nuclei of cells assuming many varieties of size and shape. These changes are attributed to nuclear division. The cord showed gliosis throughout with atypical forms of rod cells.

Glial fiber formation was remarkable in the border of the cortex, around vessels forming an extraordinary protecting wall. In the region of the internal capsule and lenticular nucleus on the left side glial fibers occupied the entire area. The brain vessels were sclerotic, alterations being most marked in the circumscribed areas of the right side, but there was withal, insufficient evidence that the vessel changes were primary. The Purkinje fibers of the cerebellum were found sometimes in the molecular layer (a heterotopia), and the processes were much swollen.

The author discusses the differential diagnosis as between glioma and diffuse sclerosis, having ruled out paresis by the serologic tests and pathologic changes found. As to etiology, he concludes, on the basis of the hypoplastic conditions, that the underlying factor is congenital and that the proliferative process is primary, and the condition a true diffuse sclerosis.

Patten, Philadelphia.

LETHARGIC ENCEPHALITIS: SYMPTOMATOLOGY AND HISTOPATHOLOGY. E. M. HAMMES and J. C. MCKINLEY, Arch. Int. Med. **26**:1 (July) 1920.

The authors report observations in twenty-seven cases of lethargic encephalitis which followed the influenza epidemic of 1919-1920. The chief clinical manifestations noted were: gradual onset with severe and diffuse headaches, becoming persistent, asthenia, lethargy, muscular rigidity with masklike features, cranial nerve palsies — most frequently the third and sixth nerves — pupillary changes, temperature ranging from normal to 103.4 F., mental confusion—sometimes resembling one or another of the psychoses—persistency of the lethargy from weeks to months, and indefinitely.

The laboratory findings were quite uniform: blood, mild leukocytosis; spinal fluid, mild lymphocytosis (normal to 57 cells per c.mm.) and mild globulin reactions.

The pathologic and histologic changes varied somewhat, but in the main the general picture was that of congestion and edema, petechial hemorrhages, pigmentation, perivascular infiltration of round cells, proliferative changes in the endothelial and interstitial tissues, and degenerative changes in nerve cells and myelin sheaths. The entire nervous system showed involvement, but most marked changes occurred in the basal ganglions, midbrain, pons and medulla. Gross evidence of encephalitis was lacking except for congestion of meningeal vessels. The predominant cell in the brain tissue was the lymphocyte, but a few plasma cells were present. There was, in addition, some increase in the glial nuclei and satellitosis was fairly common. Around the root of the fifth nerve in one case there was marked perivascular and diffuse infiltration of small round cells and hemorrhage into the subarachnoid space; there were also found in this and other cases mononucleated cells which were undoubtedly actively phagocytic and probably of endothelial origin. All nerve cells in the areas of principal involvement showed evidence of some type of degeneration and marked pigmentation, the pigment filling the cell and by its staining reaction demonstrating a lipochrome material; this pigment was also found in the surrounding interstitial tissue. Chromatolysis was frequently observed.

The etiology of lethargic encephalitis was not established by the study of these cases, but the suggestive clinical relationship to influenza is emphasized. Note is also made of the profound general symptomatology and the less marked localizing symptoms.

PATTEN, Philadelphia.

MANIFESTATIONS OCULAIRES DE L'ENCEPHALITE LETHARGIQUE (OCULAR MANIFESTATIONS OF LETHARGIC ENCEPHALITIS). F. DE LAPERSONNE, Presse méd. **28**:493 (July 21) 1920.

According to Lapersonne, the incidence of ocular symptoms in cases of lethargic encephalitis will be found much greater than 70-75 per cent., the estimate of Achard and Netter. He calls attention to the considerable number of ambulatory cases that are seen only by oculists, and to another group, not usually seen by oculists, in which palsies are not demonstrable in an ordinary neurologic examination but can be brought out by finer ophthalmologic tests. Aside from the common ptosis, he finds that internal rectus paralyses and uncomplicated paralyses of accommodation are frequent and often missed. Uncomplicated fourth nerve affections have not been reported.

Pathologic findings in the mesencephalon, suggestive of very frequent involvement of eye muscle function, are quoted from P. Marie and Tretiakoff, and from Lhermitte and Saint-Martin, who called the disease a "primary poliomesencephalitis with narcolepsy."

In general, the eye muscle palsies of lethargic encephalitis are noteworthy for their tendency to be dissociated, partial, fleeting, migratory and relapsing. A real myasthenic condition, rather than simple motor paralysis, would best explain the incompleteness, the absence of reaction (as forehead wrinkling in cases of ptosis), and the almost daily modifications. Another prominent characteristic is a tendency for ocular symptoms to persist after all others have disappeared.

Huddleson, New York.

TETANOS. FORMES CLINIQUES. TETANOS CEPHALIQUE. SEROTHERAPIE (TETANUS—CLINICAL FORMS. CEPHALIC TETANUS. SERUM THERAPY). C. Achard, Progrès méd. **35**:325 (July 24) 1920.

The author describes the clinical features of generalized tetanus and those special types of tetanus in which the contractures are localized; for example, there is a monoplegic form, a paraplegic form and an abdominal thoracic form. Of all the localized forms, the least rare but the best studied is the cephalic form. In it, apart from the head, the neurologic status is negative. Even neck rigidity is absent.

Cephalic tetanus is characterized by headache, fever, trismus and paralysis, usually of the facial muscles. It is generally occasioned by a wound of the forehead or other part of the face or head. It is made conspicuous by the mingling in close association of paralytic and contracture phenomena. The facial paralysis, often unilateral, or if bilateral, unequal in intensity on the two sides, is peripheral in type. Intrapetrosal localization was present in only one reported case. In that case, there were hyperacusis and disturbance of taste limited to the paralyzed side.

Facial paralysis is not the only paralysis seen in cephalic tetanus. Ophthalmoplegias, partial or complete, are sometimes encountered. In exceptional cases, hypoglossal paralysis is added to the facial ophthalmoplegic syndrome. It would appear that with these somewhat varied paralytic possibilities trismus must of necessity be present in order to permit diagnosis.

The writer mentions that the pathogeny of these special types of tetanus is far from clear and concludes with a discussion of the prognosis in tetanus in general and a brief summary concerning its treatment with serum.

Davis, New York.

ACUTE CEREBROCEREBELLAR ATAXIA. J. P. Crozer Griffith, Am. J. Dis. Child. **20**:2 (Aug.) 1920.

This is a report of three new cases and a summary of one case, reported by the author five years before, of encephalitis occurring in children with predominent involvement of the cerebellum but also with cerebral signs and symptoms. In all the cases the condition came on fairly acutely, with stupor occurring in only one case (in which the author himself considers the diagnosis of lethargic encephalitis) with marked ataxia, unilateral or bilateral; with speech disturbance, which the author thinks might be due to "an ataxia of speech rather than a mental disturbance;" with disturbance of mentality; with nystagmus in only two of the cases and with an absence of vertigo.

The after-results are rather interesting: The first patient recovered absolutely; the second had incoordination persisting for more than three years after the attack; the third suffered nine months later slight mental retardation and some cerebellar symptoms; and the fourth recovered after four months. The case reported by Taylor, in 1904, is mentioned in which there were some symptoms three and a half years after the attack, but none twenty years later. The collected results from the seventeen previous cases reported shows that in the majority of cases the disease leaves no traces.

Winkelman, Philadelphia.

NOUVELLES RECHERCHES SUR LA CIRCULATION DU LIQUIDE CEPHALORACHIDIEN (NEW STUDIES OF THE CEREBROSPINAL FLUID CIRCULATION). V. Stepleanu-Horbatsky, Presse méd. **28**:254 (April 28) 1920.

On the basis of these studies, the experimenter concludes that the subarachnoid space with its ramifications constitutes a lymphatic system for central and peripheral nerve tissue. He finds evidence for three normal directions of flow in the cerebrospinal fluid: primarily, from the subarachnoid space through its continuation into perineural spaces, to lymphatics of the nerve sheaths, and thence by the usual lymphatic channels to the venous system; secondarily, directly into the venous circulation of the brain; and again, but apparently not always, into the lymphatics of blood vessel sheaths, and through the lymphatic to the venous system. The fluid gradually spreads from the center peripherally, and no evidence was found indicating a complete cycle of circulation.

Subarachnoid injections of Prussian blue in cadavers were used to demonstrate circulatory channels, but the chief interest of the paper lies in the verification and amplification of such findings by means of experiments on moribund infants. Each of four patients, ranging from 9 months to 11 years of age, was given a subarachnoid injection of 1 c.c. of a 5 per cent. solution of methylene blue by lumbar puncture. Necropsies were made from ten to forty-eight hours later.

Huddleson, New York.

PERIPHERAL NERVE INJURIES. J. F. Corbett, Minnesota Med. **3**:422, 1920.

The author states that from 60 to 80 per cent. of patients with nerve injuries from war wounds recover with physiotherapy alone; the remaining 20 to 40 per cent. require surgery. If the condition is stationary for three months after the wound has healed, operation is indicated. Corbett then discusses the causes of so-called physiologic and anatomic interruption. In his case 102 days after healing was the average time of beginning of voluntary motion in paralysis of the musculospiral. In order to obtain spontaneous or postoperative recovery proper splinting is necessary.

In cases of anatomic interruption the nerve should be isolated above and below the scar, the scarred ends resected until normal fasciculi are encountered, the scar tissue from adjacent structures removed, and the ends of the nerve sutured with from three to seven interrupted sutures of fine linen or silk. Liquid petrolatum is placed about the suture line. Defects are corrected by posture of the limb or by insertion of grafts; the author favors the use of autogenous sensory grafts on account of their availability. The literature on nerve grafting, nerve crossing, etc., is reviewed.

All except two of the author's patients on whom suture had been performed showed signs of improvement and promise of ultimate recovery. One patient, on whom 7.5 cm. had been resected, showed return of function in five months.

Ott, Rochester, Minn.

OCULAR NOTES ON LETHARGIC ENCEPHALITIS, WITH TWO CASE REPORTS. Gilford Dickinson, Am. J. Ophth. **3**:8 (Aug.) 1920.

The author in his article cautions against the tendency that exists in diagnosis of ocular palsies to attribute them to syphilis. He reports two cases of ocular palsies which on first impression would be diagnosed syphilitic in origin. One patient showed a definite Argyll Robertson pupil and the other had a third nerve palsy. Serologic examinations in both cases, however, were entirely negative, and later a diagnosis of mild lethargic encephalitis was made by the neurologists.

The main features pointed out in the differentiation of syphilitic ocular palsies and palsies due to other factors as in encephalitis, are the longer duration in syphilis and the tendency for the same muscles to remain involved, whereas in encephalitis the palsy shifts from one muscle or group of muscles to another, and is of comparatively short duration, the third and sixth nerves being most frequently affected.

Patten, Philadelphia.

ACUTE INFECTIOUS MYOCLONUS MULTIPLEX AND EPIDEMIC MYOCLONUS MULTIPLEX. J. Ramsay Hunt, Neurol. Bull. **2**:391 (Nov.-Dec.) 1919.

Hunt describes a special "clinical type" of disease which is characterized by lancinating pains, muscular contractions and twitchings, and a delirium of toxic origin. He cites eight cases, two of which occurred sporadically, in 1904 and 1914, respectively, and six recently. The onset in all cases was with fever and severe pains in the lower extremities, later myoclonus and myokymia, particularly of the abdominal muscles and lower limbs, and finally delirium of a toxic infectious nature. Hunt seeks to distinguish this clinical entity from other acute infectious diseases, from peripheral neuritides, from chorea, from acute poliomyelitis and from Dubini's disease. Epidemic encephalitis is considered as a probable etiologic factor.

Cases such as Hunt has described have been frequently encountered in the course of the epidemic, and numerous reports on this variety of radicular meningomyelo-encephalitis have appeared in the domestic and foreign literature. One must take exception, therefore, to the special name which he has chosen for the clinical syndrome.

Wechsler, New York.

Society Transactions

AMERICAN NEUROLOGICAL ASSOCIATION

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WOUNDS OF THE HEAD IN CIVIL LIFE. Dr. Charles L. Dana. This article appears in this issue.

DISCUSSION

Dr. Francis X. Dercum, Philadelphia, said Dr. Dana's paper applied not only to injuries of the head, but also to injuries of every portion of the body. It was a state of mind in the patient that gives rise to what is termed in the German literature "compensation hysteria." We have, unfortunately, in this country followed the method of compensation practiced in Germany. The compensation statistics in Germany were appalling before the war. What they are now of course he did not know, but in the Germany known to us previous to the war only about 9.3 per cent. of the patients with an injury presenting functional nervous troubles ever returned to work. The consequence was that compensation in Germany grew to enormous proportions. This was because of the method with which compensation was applied. Weekly sums were paid—doled out—extending, as with ourselves, over many weeks of time; and after the expiration of a certain period, about a year, the claimant had the right to apply for another examination, at which examination he was always found to be worse. As a consequence, the compensation was always raised, never lowered. At times a necessity for special forms of treatment was claimed, for which increased compensation was allowed; at other times even a special personal attendant was claimed to be necessary and the increased expense allowed. Not infrequently, it may be added, the attendant thus allowed and paid by the state proved subsequently to be a member of the family already otherwise employed.

If our attention is turned to Denmark, where the principle of a complete cash compensation, definite and final, is followed, we find that instead of only 9.3 per cent. of the patients with traumatic neuroses returning to work, 93.6 per cent. return to work. About four years ago Dr. Dercum wrote a small book on this subject, entitled "Hysteria and Accident Compensation," published by the George T. Bisel Company, Philadelphia.

The facts, Dr. Dercum repeated, were indisputable. The patient usually made a recovery from a physical injury from which he may have suffered, but the fact that compensation often extended over hundreds of weeks resulted in the fact that the claimant never recovered as long as compensation lasted. All of us have these patients with traumatic neuroses in our clinics, treat them in every possible way and yet cannot make them well. The only way to deal with them would be to change the method of compensation. It is a notorious fact that the functional nervous patients seeking damages in the courts resist every form of treatment, but that all medical treatment ceases with the settlement. The symptoms then disappear, the patient forgets all about them. He had placed a large number of such cases on record in the book just referred to.

Finally Dr. Dercum said that the method of immediate, complete and final compensation, the Denmark method, was the fairest, the kindest and the best for the injured person. The latter is paid a lump sum, and he held that the state had no right to withhold this sum in any part or to dole it out in such sums—often pitiably small—as the officials thought best. It is readily conceivable that the man who has been injured—or believes himself to have been injured—is obliged, in order to make himself a fit subject for compensation, to maintain his symptoms and to be ill for the specified length of time. In Denmark the extent of the injury is determined by a board of physicians, compensation is prompt and the matter concluded. The patient can invest his money or do what he pleases with it, but the significant fact is that sooner or later he returns to work.

Dr. Theodere Diller, Pittsburgh, thought that in the Government War Risk Insurance there was a better plan in operation than in the State Boards of Workingmen's Compensation. In the war risk insurance, with which he had some familiarity, the plan is something like this: A soldier who is unable to work on account of nervous symptoms following injuries in the war receives $80 a month; that is, if he is unable to work. If he is partly able to work he receives one half or one quarter of this amount.

He had examined many of these men in the last year and divided them grossly into two classes, dependent fundamentally on their temperaments, those who when possessed of severe nervous symptoms, such as Dr. Dana described, stay put and the others who look forward. There are those who look backward and within, and those who look forward and without. These groups run into each other very much, but still it is rather useful to make this broad division. The ones that look forward can be helped a great deal, and the government has a good way of doing it. If a man says that he cannot work, that he cannot do this or that, the government says, "All right, we will give you lighter work." For instance, the man who complains that his work as a carpenter is too much for him is sent into a greenhouse, or he takes up some other light work, and is put on to harder work as he is able to do it. With encouragement, there is a great deal that can be done in that way. With the other class—those who look in—it is hardly possible to accomplish anything. They are always complaining that it is the government who has done this and has caused all this and shall be made to pay for it. It is hard to do anything with that fundamental condition of temperament. Dr. Diller said that giving a man a little work to do and encouraging him to do more would prove as valuable in the cases mentioned by Dr. Dana as it has proved in the government's cases.

Dr. Harvey Cushing, Boston, said he felt much troubled and disturbed by Dr. Dana's paper on this subject. He felt that we know very little about these cases. He did not believe that these symptoms occurred only in people engaged in litigation. It may have appeared so before the war, partly because most people who have injuries of the head (and it is those injuries we are speaking of, not injuries in general) were usually of the laboring classes, and not intellectual people. On the other hand, we must remember that people with such injuries who have intelligence often do not persist in consulting physicians because they see it does no good. However this may be, poor people with the complaints we designate as post-traumatic neuroses formerly appeared to be the more numerous. But the war has changed all this. He thought that a cerebral injury, no matter in what walk of life the person may be, may produce these symptoms. Soldiers who have been victims of cerebral injuries represent, in the long run, men who are not afflicted with litigation neurosis. They are seriously damaged by these injuries. They are incapable of carrying on their former work, even though there are no localizing symptoms. He did not see why Dr. Dana felt that if you cannot determine some definite local lesion, there had been no damage. These unfortunate people are left with disturbances in mentality which affect their memory, disposition and powers of concentration. They are in consequence often unable to engage in their former occupation. It may be called a functional neurosis, but it is a very definite thing. He did not think we could smooth this over, or excuse ourselves by saying that these people must not have compensation because they will not go to work. Most of these patients whom he saw were eager to go to work and made frequent attempts to do so, but they broke down under any mental strain.

Dr. Cushing had visited a hospital in England where anywhere from 200 to 300 patients, who had not been operated on, were lying, and the attendant asked him what could be done. The patients had headaches, vertigo, a sense of incapacity, and were unable to carry on their occupation. They were fairly comfortable if left in bed. They should have been operated on at the time of the injury, and that was the result. Then he had gone to another hospital and had been asked again, "What shall we do? These people have been operated on, but have holes left in their skulls. They are subject to headaches, have dizziness, vertigo, mental confusion and the old familiar traumatic neurosis syndrome. The trouble is that they have been operated on, and we must now close the cranial defects left in their heads."

Dr. Cushing said that all these people had had a serious damage to their central nervous system, and he believed that they were just as badly crippled as if they had lost their legs. The people who had lost their legs get the compensation, and the people with serious mental disturbances do not. It is the kind of disability one cannot weigh or measure by ordinary standards, but it is a very real thing. Unquestionably it is a condition easily simulated. It is a state, moreover, on which a marked functional superstructure of symptoms may develop. Nevertheless, a large proportion of the victims of serious cerebral contusions are permanently handicapped and must pursue their former occupation with lessened intellectual vigor and staying powers.

Dr. Edward B. Angell, Rochester, said that he had a good deal of success in the treatment of some of these cases with a very simple method. For those who were interested in the matter sufficiently to follow it out, he thought that one would be able to clear up a large majority of psychasthenic or neurasthenic patients in this manner. If the urine of such patients is placed over nitric acid, a pigment ring will result. When you have this with the other chain of symptoms, it means that there is a depressed tone of the mind. Analysis of the neurologic symptoms will show that along with this depressed tone there are disturbances of metabolism, usually due to toxemia. By clearing up that pigment ring one will find that the patients forget their troubles. This can be done with plenty of bicarbonate of soda between meals and sodium salicylate in 10 grain doses after meals. It is a simple method and usually efficient.

Dr. Dana said that these people have not much subconscious mind. On the contrary, Dr. Angell believed that it was all the mind they had. They had no reasoning power, for rational argument was of little avail in controlling the neurosis.

Dr. Bernard Sachs, New York, said his sympathy was almost entirely with the attitude taken by Dr. Dana and he wanted to express his dissent from the position assumed by Dr. Cushing. He did not think Dr. Cushing realized what kind of cases they had to deal with in New York, where the situation was extremely serious, and demanded serious attention. He felt that neurologists should stand together and adopt some uniform policy, particularly with reference to court cases.

Dr. Cushing was right in that there is a certain amount of injury in brain cases which is tangible—not intangible—which has been done to the brain. These are not the cases, however, which Dr. Dana had in mind, and the cases on which some immediate decision had to be taken. Dr. Sachs said there was a large number of persons who received slight injuries of the head, but from the fact that the injury had been to the head (and most people seem to appreciate that part of the body more than any other) they at once became imbued with the idea that some serious injury had been done. They constituted a large proportion of head injuries which were dealt with in court before the State Industrial Commission.

Dr. Sachs had tried to be just and to assure adequate compensation to the workingman and to play fair with him; but he admitted that the impression remained that the workingman's apparent psychosis or mental attitude was due first of all to the intense fear that he was not going to be able to continue to earn his livelihood. That was the predominant factor in his mental make-up for the time being. Dr. Sachs had not been able to escape the conviction that in the vast majority of cases it was the desire for compensation. He thought the common sense attitude of all neurologists toward this will have to be that it is the desire of compensation that often prevents these people from getting well. He thought that it should be recommended that compensation should depend on a return to work. He had appeared before the State Industrial Commission and presented that point of view, that is, to induce the workingmen to return to work and tell them that their compensation would be increased if they returned. He wanted to get away from any maudlin sentimentality that we were unfair. We are all prone to do all we possibly can in favor of the workingman, but unless he returned to work of one kind or another, Dr. Sachs feared we would have a large number of idlers on our hands for many years to come.

Dr. Foster Kennedy, New York City, asked Dr. Cushing on what authority he stated that all people who had head injuries and had symptoms afterward had vasomotor disturbances. It did not seem to him to be a scientific statement, and he would like to have the evidence. Dr. Cushing seemed to neglect the evidence that injury rendered a man liable to suggestion, and it was this that precipitated many of the symptoms and perpetuated them.

Dr. Kennedy further said that Dr. Cushing perhaps had noticed some of the reports brought out by Wilfred Trotter, who had treated cases of bone defects of the skull by taking bone grafts from the tibia; and entirely cured the bone defect. These men, both privates and officers, had serious subjective physical sensations before operation. Trotter stated that nothing was done to the brain; the bone defect merely was corrected and he believed and justly that the correction of the bone defect produced such a suggestion of cure as to cause the men to improve.

The conduct disorder mentioned by Dr. Dana lies deeply seated in our civilization and is attributable to the fact that under the industrial system the average workingman does not like his work, and is very glad to find a way of escape from toil in which he has no objective beyond personal freedom from hunger and the propagation of his kind.

Dr. Colin K. Russel, Montreal, said that we all know that there are some sincere cases among this class of what Dr. Dana has called "conduct disorders." We are also aware of the fact that a certain percentage are, as expressed in the army, "lead swingers," and each case has to be worked out on its individual merits, and it is only with time and patience that these cases can be differentiated. It is much easier to give a patient the favor of the doubt and say he is sincere than to accuse him of "lead swinging." But there was one constructive idea in Dr. Dana's paper of which he was strongly in favor. We ought to profit by the experience we have had in the army. Perhaps physicians with the British and French Armies appreciate this particular point more than those who have served with the American forces. These physicians went along for a year or more, almost two years, without any specialized hospital to look after these psychoneurotics, and saw the results and could compare them with those obtained after special shell-shock hospitals were established. He had no doubt, if Dr. Dana's idea could be realized and these persons could receive special attention in the early stages of their disability, that there would be a great saving to all concerned.

Dr. Charles L. Dana, in closing, said he was glad that this subject had aroused so much interest. He hoped it would be taken up by the neurologic societies of the country, so that more attention would be paid to legislation regarding compensation methods, and also so that more definite views on the subject might be secured from neurologists.

In regard to Dr. Cushing's statements, he believed that he was correct to this extent that not all of these patients recover when compensation is given.

Dr. Dana thought that what we lack is a definite knowledge of the natural history of this particular group of injuries. He could not find any complete description of them. A bruise of the brain is not like an ordinary injury.

THE CONTROL OF INTRACTABLE PAIN BY SECTION OF THE ANTEROLATERAL COLUMNS OF THE CORD. Dr. Charles H. Frazier. This article appeared in the August issue, page 137.

DISCUSSION

Dr. Adolf Meyer, Baltimore, asked Dr. Frazier and Dr. Spiller whether, in these operations, they had found any evidence of the existence of a bundle claimed by Head and designated as Tractus spinothalamicus ventralis for touch—a bundle of fibers on the ventral side of the ventral horn? Its existence had always seemed doubtful to Dr. Meyer, and he thought perhaps the surgical experience of these gentlemen might have thrown some light on the subject.

Dr. William G. Spiller, Philadelphia, said that the great danger in this operation was the involvement of the pyramidal tract, because if cut, paralysis would result. It is not an operation which a surgeon should undertake, unless he has had considerable experience in the surgery of the spinal cord, because it is an exceedingly delicate one. If the pyramidal tract were damaged slightly, as was probably the case in the patient referred to by Dr. Frazier, permanent disability might not exist, but it is not an agreeable thought that one has been the cause of producing paralysis in the lower limb of a man who had no paralysis before operation.

He did not believe the fibers of pain and temperature were situated in Gowers' tract, but that they lie rather interiorly, and that often the surgeon did not cut a sufficient number of these fibers to produce the loss of sensations of pain and temperature. Dr. Spiller could see no reason why, if this division of the cord were made in the thoracic region, a surgeon should not cut nearly to the anterior horn, and even if he were to put a part of the anterior root at the level he could not see that he would produce harm. The nerve cells in the thoracic region were not so numerous as in the cervical and lumbar regions. He did not believe (though he had not had any experience to justify the statement) that the cutting of the anterior horn in the thoracic region, or even a part of an anterior root, would produce permanent disability. In fact, one could not cut an entire anterior root, because the filaments of any one root of the thoracic segment extend over a considerable area of the spinal cord. At most, one could only cut a few of these filaments, but he must be careful to avoid the pyramidal tract. The possibility of hemorrhage must also be considered.

Dr. Charles H. Frazier, in closing, said he could not answer Dr. Meyer's question as to the section of any cord tissue on the ventral side of the ventral horn. In cutting the anterolateral columns they had not extended section beyond the ventral horn.

ON THE COINCIDENCE OF CERVICAL RIBS AND SYRINGOMYELIA.

Dr. Peter Bassoe. This article appears in this issue.

DISCUSSION

Dr. S. Philip Goodhart, New York, said that Dr. Bassoe's contribution was most important. It emphasized the need of care in diagnosis in a class of cases that was becoming more frequently recognized.

The congenital anomaly, a cervical rib in the human being, he believed could be regarded as an evidence of atavism, since in the embryo of lower vertebrates each vertebra had an attached rib. In the reptilean, the embryonic elements proceed to the formation of true ribs. Cervical rib in man was known to be not so infrequently associated with diseases of the central nervous system, and there were quite a few cases on record in which it had been found in syringomyelia.

The abnormal rib was probably only a part of the anomaly of which the defect in nerve morphology was another.

Some ten or twelve years ago he had under his observation a patient with double cervical rib, that is, one on each side. The case had been referred to him as one of progressive muscular atrophy. The diagnosis had been based on the progressive wasting of the forearm musculature. His examination revealed in addition to the muscular atrophy of the lower motor neuron type, in both forearms and hands, areas of hypesthesia and hyperalgesia irregularly distributed over the left forearm. These areas varied and finally disappeared, the only permanent sensory change being an area of hypesthesia involving all forms of sensation in that part of the forearm and hand supplied by the inner cord of the brachial plexus, especially in the ulnar area. The sensory changes were found on one side only.

Fibrillary twitchings were conspicuously absent. He found two prominent osseous tumors, one just above each clavicle. The roentgen-ray examination confirmed his diagnosis of double cervical rib; a marked scoliosis was also found.

Dr. Goodhart referred the case to Dr. Alfred S. Taylor for surgical relief. Both ribs were successfully removed by Dr. Taylor and the patient was relieved of pain of which she had begun to complain, and there was no further progress of the muscular atrophy. The patient had since married twice and had almost normal use of her upper extremities. In publishing the case later, he emphasized the not uncommon association of cervical rib and syringomyelia. There was a patient with syringomyelia at the Montefiore Hospital under his care in whom pain along the inner side of the left arm was a conspicuous symptom, with marked scoliosis of the same side involving the upper dorsal and lower cervical region and great deformity; a cervical rib was demonstrated by the roentgen ray. He believed the pain in this case was due to the bony malformation with pressure on the plexus. Another peculiar anomaly in this case was underdevelopment of the ribs, all of them being very small in circumference.

The not unusual association of cervical rib with organic nervous diseases should always be borne in mind.

A PSYCHOLOGIC STUDY OF A MEDIUM. Dr. Morton Prince.

This analysis shows the birth, development and disappearance of the spirit and the origin in the medium's own subconsciousness of the knowledge given in the alleged spiritualistic communications.

DISCUSSION

Dr. William A. White, Washington, said he could add one further link to the description of the case that Dr. Prince had given. It seemed to him that there was a close connection between this compensatory mechanism and the particular incident in the past life of the medium on which she chose to base this mechanism. The old portrait that had hung in the house for many years had, as a matter of family tradition, been invested with the power of rehabilitating the family fortunes, and it was just that nucleus of wish fulfillment that hung about that whole picture which was utilized later by the patient as a mechanism for bringing about the compensatory psychosis. He thought there was an intimate connection which was quite apparent.

The presentation was exceedingly interesting and emphasized to his mind the one great and valuable communication which psychanalysis had contributed to the problem of mental disease—the fundamental conception of psychologic determinism. Dr. Prince was able to work out this psychologic fancy because he approached it with the firm conviction that psychologic facts were just as much facts as any other kind of experimental facts, and that they were not incidental chance, but that there must be adequate cause for them, and that cause might be determined as a psychologic one—that they must have some adequate goal toward which they were reaching. With a firm conviction of psychologic determinism, psychologic fact and psychologic causes, and that they existed for a purpose, these cases could all be analyzed just as clearly as Dr. Prince had analyzed this case.

Dr. Morton Prince, in closing, said that in narrating the analysis of this case of course he had left out a great many details and especially those which concerned the motive. Undoubtedly there was a strong desire to rehabilitate the family fortunes, but he thought the main motive was based on the promise made to her for the attainment of future happiness. He also thought there was a side to her character, although repudiated by the dominant side, which possessed a love of notoriety. The main motive, however, was the desire for happiness. He should have pointed out, as it was a matter of interest, that after the analysis had been made and the real facts had been brought to light, she said—referring to spiritualism and her supposed mediumship—"I have had enough of them: I do not want to hear about anything more or investigate further the alleged spirits; I am quite satisfied." After the revelations she was for a brief time distressed beyond measure and for the moment fell back into the state of her former depression. This was due to a realization of the false position in which she had placed herself because of having deceived, although unwittingly, herself and, above all, her friends who had had such trust and confidence in her. This state of mind, however, was one Dr. Prince easily corrected.

A DIAGNOSTIC CLINIC FOR ADOLESCENTS: PURPOSE AND ORGANIZATION. Dr. Pearce Bailey.

A number of years ago, in considering what psychiatric and social diagnosis could do for education, Dr. Bailey was struck by the large number of people who early in their careers gave signs of imperfect adjustment to their environment. This was shown by military statistics which in some countries, such as Germany, showed nearly 50 per cent. of recruits to be unfit for first class military service. It was shown in this country by statistics dealing with higher education. For example, in one of our schools of technology not more than forty out of each hundred entrants receive diplomas at the end of four years. Statistics from schools that maintain a follow-up system of their graduates showed that 20 per cent. of the students who entered did not graduate from college. In all large schools for boys and girls there are constantly pupils who do not fit in, and the parents or teachers are not able to find out just why.

The misfits and failures in occupational and social relations during adolescence, although more difficult to determine statistically, are too well recognized to make it necessary to take time to dilate on them before this Association. They concern boys and girls both, but since boys are, or perhaps it is safer to say have been, up till now, more important as economic assets, their careers more in view, the failures of boys have received more attention than have those of girls. It is boys on whom money is spent, for whom outlay is made, either as commercial backing or as advanced education, and so when investments of this kind turn out badly, it is boys that we hear talked about more.

Adolescent inadequacy has been recognized for years, but it required the draft examinations to disclose how widespread it is, and even now it is doubtful that there are many prepared to believe, in spite of the proofs, that more than 30 per cent. of American adolescents are handicapped in some way and require some building up, some adjustment, some special allowances and facilities before they can hope to attain all that they might attain under favorable circumstances.

The medical profession is naturally consulted in matters of this kind, and it stands ready, as it always has, to render service, paid for inadequately or not at all, for public improvement. But of this particular problem it has yet touched only the fringe. It has gone as far as aldermen and legislators permit. In the best city schools some attempt is made to classify pupils on the basis of physical and mental status, and here and there psychiatric clinics have sprung up, designed chiefly for the benefit of those actually ill, or of those whose inadequacy has manifested itself in clashes with law and order. Most of the work so far done concerns mental defectives, the insane, neurotics and delinquents. As far as I know, there exists nowhere a personality clinic—a clinic for the estimation of a handicapped person's weak and strong points, based on complete psychologic and physical investigation, with the purpose of pointing out the most favorable adjustment for him.

The organization of such a clinic encounters great practical difficulties on the score of expense. To examine a person so as to really know him, requires a great deal of time, a number of highly experienced examiners, to say nothing of the outlay involved in laboratory work, roentgen ray, etc. With the present lack of appreciation of the importance of such work, it is imposible to rely on public money to finance it; neither will the great philanthropic foundations, as at present administered, stand behind such an undertaking. As things are at present a thorough diagnostic clinic for adolescents, or classification clinic as it may more appropriately be called, would have to be organized on the basis of largely or entirely supporting itself. It would do this by charging fees large enough to meet the running expenses.

Such a plan of organization carries with it certain practical advantages. In the first place, it furnishes at once a means of selection of candidates who are to be examined. A clinic whose avowed purpose was to prevent or compensate for failures, and which offered free services, would be at once over-run with applications, and would have no means of selecting among them. On a pay basis applications would come only from those parents or guardians who were contemplating investment, educationally, commercially or otherwise on the adolescent, and who really desired expert advice before doing so. This would mean that such a clinic would deal with a provident class, which would in itself have an important bearing on the value of its final statistics. The examinations should constitute a complete record of the candidate as an individual at the time the examinations were made. This record should show the results of heredity, of early surroundings and education, together with the demonstration of mental or physical limitations, as well as the existence of special talents or aptitudes. It would be a personality summary at a certain period of the individual's career, which would only acquire scientific value of importance if checked up, later on, by information as to what happened in following years to an adolescent who at a certain period of his development had a certain static record. It is probable that a clinic which dealt with a provident class could carry on a successful follow-up system through long periods, which is impossible with the types of adolescents that frequent free clinics. It seems that such a follow-up system could not fail to furnish facts, now almost totally wanting, invaluable to the understanding of the evolution of character. It would make available knowledge, not only as to the prospects of an adolescent with a certain personality status, but also the conditions that would have to be fulfilled in order that he might find the best adjustment.

The Neurological Institute of New York has long maintained as a part of its outpatient department, a clinic in which difficult and exceptional children have been studied. The cases have been largely drawn from the public schools. It proposes, on Oct. 1, 1920, to supplement this activity by the establishment of a pay clinic which would receive adolescents not necessarily "pathologic," and in which the examinations will be more thorough and more extensive than is possible in a free clinic. They will be made irrespective of any symptoms complained of, and will attempt to establish variations from the usual as well as pathologic symptoms. The work will be conducted by the regular staff of the institute supplemented by such additional assistance as may prove necessary. When the examinations are completed an opinion and recommendations will be furnished to the physician or whoever referred the case. The clinic will not undertake treatment of any kind.

DISCUSSION

Miss Elizabeth Farrell, New York, by invitation, said it was a particular pleasure for a teacher or school administrator to hear from a neurologist the words that Dr. Bailey had spoken. In New York they would welcome the organization of any clinic in a way that Dr. Bailey himself did not appreciate.

Her own work began with backward children and defectives, but she would dismiss that whole subject with the statement that they had more than 4,000 of these children in their classes today.

The problem which was uppermost in their minds was that of children not mentally defective, per se, but of children who presented problems of behavior which finally worked out into real social problems, after school life, in the community and in the world. If she could add anything to Dr. Bailey's suggestion it would be that this clinic get the child early in order that they might get what Dr. White told them always to get, namely, those early manifestations of maladjustment which, if not corrected, finally evolve into serious maladjustments of one kind or another.

These children may be divided into two groups—those who are highly gifted and superior children, and those children who are not so gifted. The first group represents the most serious problem in school administration that must be dealt with now—what is to be done with them and how is it to be done? The gifted children are the most neglected of all the children in the schools today. That is true of the schools in Pittsburgh, and it is true as well of all schools all over the country. She thought the fault was that society demanded of schools a certain uniformity of teaching and of subject matter that was deadening, and that it was the maker of mischief and of problems in children who were different from other children. She stated that these gifted children were not worked to their full capacity, and developed very real behavior problems. They develop a superficial attitude toward the teacher, toward the work and toward the school. They know they can do the work the school requires of them without any effort; the community is not intelligent enough to have them make the effort; and instead of getting intelligent understanding and intelligent development of these gifted children, a superficial development that makes for all sorts of deviltry and lack of achievement in later life results.

In addition to the problem of the gifted children, there is the problem of those children with special ability, and this she repeated was an indictment of all communities. They are asked to teach all children to read, write and cipher, and when wood, paints and sewing material were asked for the request was not readily granted because they were regarded as fads and frills. Her statement was an indictment of all schools. She had in mind a little boy brought up as a serious offender. He would not go to school and would do many other things. He did more harm than they thought it possible for any boy 12 years old to do, and they wondered how he could possibly have the resourcefulness to carry through some of the activities in which he had indulged. He carried them through, but was caught at last. The examination, the search, as Dr. Prince would call it, for the motives that were driving this boy on to a career chosen for himself revealed the fact that this boy, a dweller of the tenements, from a poor home, had hidden away in his personality such a gift with pencil and paper, with pen and brush, as was not found in one among thousands. The boy was not even one of the gifted children. They had of course to decide what should be done with the boy who was a great mischief maker. The ordinary procedure would have been to send him to the Truant School, where he would receive no thorough instruction along the lines of his bent. But, instead of that, he was given a chance to get training in this wonderful ability. In eight months he had developed from the undesirable character into a boy with self-respect, ability and skill, and he was interested in his career, which was a promising one.

These special ability children need to be recognized in the schools. They need to be provided for in the schools and not handled like machines, and this was a subject for consideration.

She wanted to add a word to what Dr. Bailey had said in regard to endocrinology. The number of endocrine cases that they had was perfectly astounding, and it was equally astounding to see what treatment would do for them.

Miss Farrell wanted to leave a message with the association, and that was to demand that education in communities be made to meet the capacities, the interests and the special abilities of the children.

Dr. Stewart Paton, Princeton, said he did not believe it was necessary in this Society to spend time in urging the importance of such an institution. An institution of this character not only would be of great benefit to those who went to the institution for advice, but it would also be of service to teachers who might be brought there to ask for advice about pupils. This would give the teachers a biologic conception of education. It was not necessary, however, to wait until there was such a complete organization as Dr. Bailey suggested. It was quite possible to begin now in our universities by supplementing the excellent work done by the present department of hygiene. Students should be taught the essentials of mental hygiene as part of their education.

It was amusing to reflect on the fact that while violent discussion had recently taken place in academic circles as to whether the Greek grammar should be retained in the curriculum no one seemed to think it worth while to suggest that the Greek spirit be introduced into the universities. The advice given by Socrates was that the essential part of an education was to "know thyself." It seemed to him astonishing that this point of view had received so little consideration. Dr. Paton would have liked to have shown some records of students which he had in his possession. If the heading were taken off these records, one would be unable to say whether they were students in universities, supposed to have acquired the academic essentials of education, or whether they were patients in the Phipps Clinic in Baltimore, or in Dr. Tilney's Clinic in New York. The great majority of students had not the faintest knowledge of their own adjusting capacity and limitations. As a matter of fact, there were only two or three universities in the country in which any effort ever had been made to teach mental hygiene and give students the essentials of an education. So-called educational institutions were graduating a great many maladjusted persons.

Dr. Adolf Meyer, Baltimore, said the important point in this question was the manner in which to attack the issue. Was an institution of a formal character the most desirable starting point? Would it not, perhaps, be well to keep the organization more or less in the background, and carry on the observation of the individual under the most natural conditions, instead of bringing him into a totally strange situation—that of being examined in a special institute? In this connection he emphasized what had been the upshot of some work undertaken in one of the schools of Baltimore, a school which was first made the center of investigation for the usual study of feeblemindedness, and in which spontaneously and really of necessity attention was extended to the study of the nonfeebleminded, to those who gave problems because they were too much for the teacher rather than too little. In this interesting work, carried on somewhat along the line which he described in a lecture published by Macmillan, together with lectures of Jennings, Watson and Thomas, in a book entitled "Suggestions of Modern Science Concerning Education," Dr. Richards studied the pupils about whom difficulties were reported by the teacher or by the parents or usually by both, often without any realization of the individual, though the issue was more than obtaining a greater familiarity with the pupil's problems and with his home. The point especially to be raised was that not only in the high school or university, but even in the upper grammar school stage, the important facts always referred back to the reports of the first year—the first contact the child ever had with the school. In Dr. Richards' studies it was found that there was a problem in a large proportion of those individuals from their earliest years, and therefore he urged that these investigations should be carried out in the initial stages. It should be one of the problems of the schools of today to standardize the pupils, not in the way of labeling or marking them, but in making them know what they were best fitted for. That should begin in the earliest grades and ought to be carried on from there.

As to the organization proper of the work he referred to, there had not been any special financing. The money had to come partly out of their own pockets in the beginning.

For the fundamentals, it was quite essential that one should work in the natural environment in which things happen.

Dr. Bernard Sachs, New York, stated that every one felt the need of an organization such as Dr. Bailey had so ably described, and which Miss Farrell had endorsed. He wished to present one rather practical aspect of the situation. If the community at large were to be benefited he would not advise the organization of a diagnostic clinic for the purpose of determining the mental condition or aptitudes of children. That mere fact would prevent thousands of people from coming for assistance because the implication was that by reason of that application the child was already labeled as somewhat defective. For that reason he thought it should not be called a diagnostic clinic and advised, for the present, the name of a diagnostic class. Neither did he believe it should be linked up with a hospital or institution, but with the educational system, and especially if it could have the assistance of such able women as Miss Farrell, who, in New York, had done so much in the educational system provided for defectives.

Of course, in an educational class of this kind, or in a diagnostic class of an educational system, the active support of neurologists, psychiatrists, and possibly some psychologists was necessary.

So far as the necessary financial support was concerned, he had not the slightest doubt that if the public at large were shown the benefit that would accrue to the entire community by such an organization, there would be no difficulty in getting such support. In New York City the need was great.

Dr. E. W. Taylor, Boston, said that apropos of what Dr. Sachs had just said, he would like to mention that for some years past special classes had been connected with the public school system of Boston. These classes were made up of exceptional pupils, not necessarily feebleminded, although naturally the mentally backward pupils constituted their most important element. These special classes, so far as he knew, had proved a decided success and had helped to solve the problem of the exceptional child.

Dr. Taylor gave a case history which fell into the category of that cited by Miss Farrell. It was that of a young boy, 10 or 12 years old, who came to the hospital as an incorrigible some years ago. He could no longer get on in the school, and was sent to the hospital in order that inquiry could be made into his mental state. On being questioned, he stated that what he would most like to do was to go out early in the morning, sit in a meadow and see the sun rise. On questioning him further, it was found he liked to draw, and he then made a sketch on the blackboard which was so excellent that it was preserved for several years—a train of cars coming around a curve in perspective. He was then talked to further about what he was studying in school, and it was explained to him how desirable it was, if he wanted to go to the Art School afterward, that he should study geometry and kindred subjects. He took these suggestions and became almost immediately a model pupil and did exceedingly well from that time on. Dr. Taylor had not the slightest doubt that he would be a useful citizen instead of the outcast which he was on the point of becoming when sent to the hospital for study and observation.

Dr. William Healy, Boston, stated that it was most interesting to hear a group of neurologists entering into this particular discussion. Dr. Sachs made a very good point. In developing their work in Boston, after years in Chicago, they desired to leave out the word psychopathic in order that any stigma that might be connoted by reason of this name should be avoided. There was no necessity whatever for allowing this implication to be a feature in the situation, and if it is not, one finds no trouble whatever in getting the cooperation of people in general. It was important for any one doing this type of work to see plenty of normal individuals for the sake of developing norms of a check group. They had seen cases of many school children and others who were not specifically regarded as psychopathic problems.

Just this minute it had been suggested to him by someone present that after all the discussion that had been going on showed that neurologists were not well posted on what was being done in various centers. A great deal of what had just been advocated was already being accomplished, from California to New England. In a number of communities by a variety of organizations children were being studied for problems concerning their mental make-up.

Dr. Morton Prince, Boston, said that Dr. Healy had just brought out the point he had been thinking about while the discussion had been going on, and that was whether or not, as neurologists, they were really fully equipped for attacking this big problem. He concurred with Dr. Bailey's idea, and thought there was a great deal, too, in what Dr. Sachs had said.

It had been forced on him in recent years that there were in the universities (and he had Harvard particularly in mind) a goodly number of students who were maladapted to their situation—to the demands made on them by their social and educational environment—who were unable not only to pursue satisfactorily their college courses and to fulfill their aspirations, but also to solve the everyday problems of their lives. They seemed to be misfits and a puzzle to their teachers who had undertaken to advise and guide them. He had been consulted a number of times by the teaching staff in regard to such students, but had felt inadequate to advise, partly because of the difficulty of the problems and partly because of the enormous amount of time it took to adapt such students to their situations. To do this required one who had the knowledge and could give the time to an intensive study in each individual case. Such cases involved problems of personality and he doubted very much his ability and the ability of neurologists as a class to solve such problems. They would have to know more about the personality and the conflicts that go on below and within, that is, what brought about the maladaptation, before it could be understood why these individuals were unable to adapt themselves to the conditions of their lives—to reality.

Dr. Prince thought, therefore, that it was possible that such an organization as Dr. Bailey had proposed might be begun within the colleges. It was there it was needed. And from such an organization within the colleges it might be extended to comprehend other classes of the community. He was quite sure that one of the most fruitful results of such an organization would be the education of neurologists, as well as the reeducation of these maladapted individuals. Neurologists need to be educated before they can undertake the reeducation of others.

Dr. Pearce Bailey in closing said that this idea first came to him a number of years ago in his work with Dr. Allan McLane Hamilton. He had long had the idea that the aptitudes of normal people might be determined experimentally, and that useful advice, based on the study of the normal individual, could be given as to a future career. He differed somewhat with Dr. Hamilton on that point, and his subsequent thought in the matter had referred more to the slightly subnormal.

It was easy to see that a clinic of this kind would attract, more or less, people decidedly subnormal, delinquents and mentally defective individuals. But that was not the original idea and that would not be the class that they would wish to have come to them. The idea would be to make this clinic an organization for those boys and girls who had no conspicuous defects, and yet who did not seem to be quite in harmony with their environment.

Dr. Bailey was fully cognizant with what was being done by Dr. Healy of Boston and Dr. Goddard of Ohio, but he did not take up that side of the question; for as originally conceived, this clinic was social and economic rather than psychiatric. For example, 20 per cent. of the boys who go to college have to leave. These boys are not defectives or delinquents. It was an important economic situation with their fathers. What influenced their leaving? Then there were other boys who could finish their college course who would have been much better off if their fathers had determined that they were not exactly fitted for that kind of life.

As far as the organization was concerned and Dr. Sach's remarks that it should not be connected with a hospital, of course it was necessary that a clinic of this kind should have practical support, and if this practical phase was borne in mind it made it simpler to start such a work in a hospital for the reason that the staff could be utilized so largely, because they were there and readily available. It would be an expensive procedure if organized in any other way.

NERVOUS SIGNS AND SYMPTOMS AS RELATED TO CERTAIN CAUSATIONS OF CONDUCT DISORDER. Dr. William Healy.

This article will appear in an early number.

DISCUSSION

Dr. Morton Prince, Boston, thought that the interesting paper of Dr. Healy was suggestive. He could confirm the phenomenon reported, of somatic disturbances, such as headache, pain, dizziness and nervousness, occurring at a moment previous to or during the eruption into the mind of some impulse or of some repressed idea that had been put out of the mind. It was a phenomenon which he had observed over and over again and which was quite common in certain types of cases. He had never had any experience in the kind of case — the delinquent — reported, but it was interesting to correlate the phenomenon in these cases with the same phenomenon as observed in other conditions. He would perhaps take a little different point of view in regard to the mechanism of its occurrence, to the relation of the physical phenomenon to the accompanying impulse to action or to the emergence of the idea. Investigation, in his experience, had always shown the somatic phenomenon to be due to, and the resultant of, a mental conflict—a conflict with a subconscious process. When such conflicts occurred there was apt to be a somatic symptom, such as headache, dizziness, etc. The latter stood in a resultant, not a causal relation to the conflict, that is, to an idea with its impulse striving to obtain expression.

In the very case he reported he had seen on a number of occasions the production of these somatic symptoms when a subconscious motive, or impulse or mental system was endeavoring to come to the surface and obtain expression. On one occasion, for instance, the patient complained of intense headache. This was easily traced, as he anticipated it would be, to a mental conflict—to ideas which had been repressed and which were attempting to determine the conduct of the personality—to erupt into consciousness. In the course of the interview, as soon as the conflict was relieved, the headache immediately ceased. On another occasion there was intense headache, nausea and dizziness. These immediately ceased on the solution of the conflict when the subject was hypnotized. She then recognized herself that these somatic symptoms were only the result of the conflict and were not due to any physical illness, as she had thought was the case during the time of their occurrence. In another patient he had seen again and again what were apparently sick headaches, for which her attending physicians had given morphin and every sort of medicine without relief. In that case it was easy to trace the headache to mental conflicts which were correspondingly easily and immediately dissipated by relieving the mental condition.

While in such instances these somatic symptoms may be interpreted as an attempt at a defense reaction, yet from another point of view, and especially in certain cases, he thought they might be interpreted as being due to the discharge of emotion along sensory pathways.

After all, this phenomenon, it seemed to him, was observed in normal individuals in the course of everyday life. Given a person with a certain kind of temperament and let that person be torn between two desires or impulses, which meant a conflict which he could not for the moment satisfactorily solve for himself, and he would complain of headache, nervousness, dizziness, etc., resulting from the struggle and antagonism between the two impulses. This was the common, everyday phenomenon which we had all observed and which perhaps some of us had experienced. The principle, it seemed to him, was the same as in abnormal cases.

It must not be inferred from anything that he had said that every time one sees a person with a headache, or other nervous manifestations to which reference had been made, that it is necessary to seek for a conflict of some kind, or that there is an attempt of some subconscious impulse, or idea to erupt into consciousness. The same kind of somatic symptoms occurred, of course, under all sorts of conditions.

Dr. Smith Ely Jelliffe, New York, said that the clinical material which Dr. Healy had presented seemed to him to afford an extremely interesting and suggestive clue to the beginning of a type of processes, to which Dr. Prince had just referred, some of which had interested him for a great many years.

He reminded the Association of a photograph in the "Scarlet Letter," in which Hawthorne says, "that a bodily disease which we think of as a thing apart and separate in itself, may be after all only a symptom of some disturbance in the spiritual part of our nature." Hawthorne had the right idea, and Dr. Jelliffe was convinced that Dr. Healy had given an excellent picture of the beginning of this split, whereby the body was made to become the scapegoat of the spirit. It must be assumed in the process of evolution that ethical imperatives biologically implanted for thousands, perhaps millions of years, caused a striving toward better social adjustment of the individual. These young juvenile offenders showed in the minor physical or somatic symptoms, the results of the conflict between their inherited biologic ethical imperatives and the conditioned reflexes of individual self satisfaction. They compromised, partially carried out the behavior of pleasurable gratification, but not with complete somatic success. The body symptoms showed that the ethical imperative, or what the religious dialectic would term, conscience, had a hand in the compromise reaction, and thus to recur to Hawthorne's line, the bodily discomfort was the symptom of the spiritual maladjustment.

To Dr. Jelliffe the important suggestion was that whereas Dr. Healy had pointed out some of these minor manifestations of a conflict with a satisfaction of the opposing trends, or a partial satisfaction of the opposing trends —with some stealing and some somatic disturbances—as these patients were followed later in life, say fifteen, twenty, thirty, thirty-five to forty-five years, the same types of compromise going on, with different intensities, and with different types of conventional ethical milieu, they finally arrive at a group of things which we call "organic diseases," or which by specific complications as from certain infections, become special types of organic disease. And, as that was the general theme of the subject he hoped to pursue in his paper he simply emphasized the scapegoat nature of the body that wears a hair shirt, as it were, in the form of physical disease, in order that the individual might conform, in part at least, to the rulings of the herd; as it was more important for the individual to stay in the herd than for him to suffer physical disease, he tears his body down, unconsciously, in order that he may not be excluded from the herd.

Dr. Abraham Myerson, Boston, said there were a few points he would like to discuss: First, the type of conflict that Dr. Healy had brought out occurred commonly in the housewife. Many symptoms of the nervous housewife, such as headache, dizziness and vomiting, were frequently related to disloyal thoughts.

It did not seem to him that to explain this required a complicated analysis. It was not necessary to go quite so far as to say that the body was the scapegoat of the mind in order to account for the headache, dizziness and uncomfortable feelings. Any conflict of ideas had with it a group of associative somatic changes in the blood pressure, associated changes in the heart action and in the lungs and everywhere throughout the body. There were two groups of ideas and the oscillations in conflict, alternating in consciousness, between them causes such changes in the adaptive mechanism of the individual that he cannot translate them into action. The ideas themselves may be called the cerebral reverberation of events, and the associated emotion may be called bodily reverberation of events. This group of conflicting ideas cause or are associated with bodily changes that make it difficult for the individual to discharge his energy into action.

As to therapeutics, he thought that in the treatment of cases of mal adaptation to society, we forget that which we constantly keep in mind in the treatment of somatic diseases. In the latter we keep in mind that there is a natural tendency to get well. We keep in mind constantly that there are processes whereby the equilibrium is regained. It was not thought that typhoid fever was cured if calomel were prescribed and the patient recovered. More cures occurred by coincidence than through actual treatment. In the majority of cases of social maladaptation there was a natural tendency to recover, a belated adaptation that arose spontaneously. It is necessary to be as critical of our efforts in this field of therapeutics as in any other field.

Dr. Stewart Paton, Princeton, N. J., said that two cases of stealing which occurred among university students had come under his observation. There were a great many interesting points in the history of these students, but the one he wished to emphasize was that any one knowing these facts and understanding the methods of analyzing a personality would never have allowed the students to have entered a university. In one of the cases, it was easy to trace the signs of instability back to the student's earliest years.

There was a great field in our universities for any one interested in the study of the human personality. He believed it was quite possible for the Association to do a great deal of good, and he agreed with Dr. Meyer that the sooner an organization was perfected for making these examinations in the school period, the better; and the more chance there will be of introducing the biologic conception of education into academic circles.

Dr. Herman M. Adler, Chicago, asked Dr. Healy this question—What was the relation of the intelligence of Dr. Healy's patients to their behavior reactions? Because of the examinations of the soldiers in the recent war and because of our cases, it looks as though there is some relationship between the intelligence rating and these psychoneurotic manifestations. In general, our patients have shown the correlation of a high intelligence quotient with a relatively complex reaction. Where the intelligence quotient is low, the reaction is simple.

Dr. William Healy in closing said he had only to say in closing that this topic was really worthy of close attention by neurologists. Not only were many cases not being seen which should properly be studied, but cases were also frequently seen that had been passed over by neurologists who had not arrived at essential facts.

He was not altogether clear about the explanation of the results Dr. Myerson discussed. Dr. Meyerson had suggested that there might be an element of suggestion in the outcome. About that he thought one could not express oneself with surety. It may be partly this and partly due to the intrinsic value of exploration itself. For the first time somebody presented himself who was willing to listen with the right kind of interest and to make the right kind of inquiry. Where the repression and behavior reaction had taken place over years, it seemed clear that the individual had been almost waiting for this situation to present itself, waiting for an understanding listener. There was a great deal that could be said about this question of the actual cause of the wonderfully good therapeutic results that were so frequently observed, and a long discussion could be centered on this special points. The fact that these misbehaving and suffering individuals had never revealed their real inner life previously was a striking lesson for parents so rarely do anything to understand their children deeply.

The continual finding of these intricate behavior reactions only in individuals of relatively high intelligence led us to have the same opinion that Dr. Adler expressed. There seemed to be few exceptions to the principle that where the intelligence was low, the reaction in behavior could be explained in simple terms.

THE PSYCHONEUROTIC ELEMENTS IN JUVENILE DELINQUENCY.

Dr. Herman M. Adler.

Dr. Adler presented cases from the Juvenile Psychopathic Institute, Chicago, illustrating the psychoneurotic type of reaction to socially difficult situations—the conflict betweeen fear and desires. He made a comparison with forms of shell shock. He advocated therapy by suggestion and social after-care. He compared these cases with cases of adult criminals. He concluded with comments in regard to the more general application of psychiatric methods to the treatment of delinquents and criminals.

DISCUSSION

Dr. Harold N. Moyer, Chicago, said that the last case reported by Dr. Adler was one of the most bizarre that had ever come under his observation. The confession of the man was for the most part incorrect. His memory of the crime was fragmentary and confused. The case illustrated the great difficulty in sifting evidence.

Dr. Herman M. Adler, in closing, said that he read the account that the man had given. He heard from Dr. Moyer that there were some details of this sort that he was going to present, and that was the reason he selected this case. As a matter of fact, it did not make any difference. It altered a number of points, but it did not alter the essential fact that the man was amnesic and did not know what he was doing.

As a member of the state board he would like to tell Dr. Moyer that when he had all this information eventually in hand, if he would present it before the board, Dr. Moyer was sure that something would be done to commute the sentence of the man.

THE DEMENTIA PRAECOX PROBLEM. Dr. Henry A. Cotton.

Dr. Cotton presented the difficulties of diagnosis. He discussed the question whether or not dementia praecox is a disease entity; the etiology of chronic infection, and the reasons for considering dementia praecox an organic disease.

DISCUSSION

Dr. John W. Draper, New York (by invitation), said he appreciated the privilege of speaking before the Association. He knew nothing about mental conditions, but he was in a position, he believed, to say something about the surgical pathology in these cases. He and his colleague, Dr. Lynch, had operated on about 130 patients for Dr. Cotton. All the operations were abdominal, and they had the satisfaction of not opening a single abdomen without finding pathologic conditions.

It is well known that a great deal of surgery has been done on the insane —a great deal of good surgery—but it has been directed more toward the relief of pressure due to tumors and various disabilities of that type, particularly in a gynecologic way. Their work, however, had been directed chiefly toward the lesions to be found in the alimentary canal.

Dr. Draper said that the pathology present consisted in an abnormal position due to faulty development of the right side of the colon. This side of the colon is developed last; it frequently does not develop until the first year of extra-uterine life, and in many cases there is lack of fusion, and that is productive of so-called congenital bands. The result is that pathologic changes in that segment of the bowel occur later in life. He did not mean to say that this was the only segment of the bowel that had been involved in the cases which Dr. Cotton had presented for study, because in a great many there was also involvement of the oral portion of the small intestine. However, there is no doubt that the right side of the large bowel is the decadent portion of the alimentary canal, probably owing to its late acquisition. At all events, whatever the cause, the pathology is there.

Of course, they had examined the specimens that had been removed, that is, anywhere from 18 inches to 2 or 3 feet of the segment involving the terminal ileum, cecum, ascending and transverse colons. The terminal ileum arose from the hind intestine and participated with the cecum in this tendency to infection. Dr. Ewing and other pathologists, had reported that these specimens contained as much tissue change as they had ever seen, Everything was involved. In many of them there was an immense increase in the fibrous tissue, a general fibrosis, in which the blood vessels, of course, shared. Most particular and important of all, there was an invariable involvement of the mesenteric glands. They had removed many hundreds of these glands at operation, and if cultivated, they always yielded living organisms—streptococcus and colon bacilli of varying types being the most constant.

Dr. Ernest Sachs, St. Louis, stated that as a one time general surgeon and as a teacher of surgical pathology for the last ten years, he would like to register a remonstrance against the surgical work in the right iliac fossa in these cases. The best monograph he knew of on studies of the appendix, which was recognized all over the world, was that by Professor Aschoff of Freiburg, who showed that there is no appendix in any one above the age of 3 or 4 which did not show the pathologic changes that Dr. Draper had just described. In other words, one never sees in an adult a normal appendix—one in which these changes do not occur in the musculature, fragmentation, infiltration of leukocytes occasionally, and, of course, abnormal position. This gastro-enterologists doing a great deal of roentgen-ray work had recognized for a long time.

Dr. Sachs said we had just recovered from the epidemic of removal of the colon for epilepsy which started in Cincinnati. He had the privilege of seeing some of these cases before and after operation. It may not be known to all that in a public meeting in Cincinnati Dr. Reed was obliged to retract all that he had claimed for this operation on epileptics.

Dr. Sachs felt strongly that there was nothing that was going to bring surgery into disrepute as much as this type of surgical work, because he thought it had been shown beyond all question that the various configurations of the large intestine and those of the other intestinal organs vary enormously in different types, and most surgeons feel quite certain that they can place little faith in the work first popularized by Lane of London.

Dr. Charles K. Mills, Philadelphia, said that he had elsewhere presented his views with regard to certain phases of this subject. He had no doubt that the remarks of Dr. Sachs were entirely correct and applicable. The essential thing was that we should not be misled with regard to the etiology of well-known mental diseases by these reports on the discovery and removal of infections, whether in the teeth, the tonsils, the intestines or wherever the location may be. It was undoubtedly true that the idea had gone out that dementia praecox, manic depressive insanity and ordinary paranoia were due to infection and could be cured by the removal of the infection. He did not believe that there was any truth in this statement. It would be difficult in a few words to say what these affections were. Perhaps the word teratologic or embryonal expressed the idea of their nature in a general way.

Unquestionably, insane patients often had bad teeth, but so had multitudes of others who were not insane. But, as he read the title of this paper, it was "The Dementia Praecox Problem." The problem of dementia praecox, as presented here today, would seem to indicate that the essential cause of this disease was infection, which as he had said he did not believe was true. Many good teeth had been removed because through the newspapers, as well as through other publications, the idea had reached the public mind that a common cause of mental trouble was to be found in diseased teeth.

Dr. Adolf Meyer, Baltimore, said that this was a difficult problem to discuss unless we dissolved it into the component factors. Unless we got to the point where we are perfectly willing to discuss honestly and sincerely the independent factors before we speculate about "the pathology" found in the intestinal tract and the diagnosis of dementia praecox, we cannot regard ourselves as intellectually safe. He admitted a certain suspicion when he heard any one speak of "finding a great deal of pathology" in the intestinal tract. Disorders or lesions may be found, and yet we may have a hard time to determine what "pathology" would explain these facts.

It seemed absolutely essential to recognize that a multicellular individual, such as man, an individual with so many organs, an individual with so many functions, and an individual with so many problems to meet, could not be dealt with under just one category, by giving an array of pictures of teeth and speaking about the mental problems as if they were merely incidental. It was wrong to discuss mental pictures leaving organic difficulties out of the question. It seemed to him that if we wish to attack this problem on its proper basis it was essential that we cultivate a certain type of intellectual morality, accepting the fact that the individual was a complex mechanism made up of many factors; that we did well to deal with the different factors independently until we knew exactly what they were; and that then we might begin to show what their correlation may be. From this point of view, Dr. Meyer had relatively little patience with those who scoff at a more careful study of the gastro-intestinal tract and of the infections, whatever they might be. A careful study should be made of them. It was not an adequate excuse for not taking up a thorough investigation of disorders of the intestinal tract simply to say: "Nearly everybody has them." The fact that many people were able to drink alcohol with impunity, did not change the fact that with some persons its use was disastrous. It was natural, of course, that we should use our judgment with regard to the extent to which we should go into special factors. We realized that we could not all do everything and we therefore focused on the things we could do best, and on that which was likely to mean most for the patient.

For the sake of research, his suggestion would be that some of the foundations that lavishly support many other directions of investigation, might perhaps be induced to put aside some funds for an unquestionably unbiased corps of workers who would take up the various essential aspects of this problem and carry the work through as thoroughly as it could be done. To do that would take a great deal of work, and above all people of great experience. It certainly could not be done out of nothing or with the merely partial organizations of today. If there was any possibility of inducing some of our foundations to take a positive interest in this we should have a much more instructive and profitable time than with a discussion of the question, which gives us only one or two aspects of the problems in each patient.

Dr. William A. Jones, Minneapolis, said that it seemed to him that all this discussion was like "a tempest in a thermos bottle." Dr. Cotton, in presenting his subject, based his studies and his conclusions on a sufficient number of cases from which to draw a lesson. The pith of his paper was that each patient should be given a complete and thorough examination and, also, any pathologic condition that might influence the future life of the individual should be removed. Whatever the conclusions were, Dr. Cotton's methods of investigation were good and sound.

Dr. Henry A. Cotton, in closing, said that of course one could not give all of one's data in the time allowed for reading a paper, and in the remaining few minutes he wanted to give some of the results. He thought that it would be admitted that twenty years in this work had given him a little experience. When he said that for eighteen years he did not do anything for his patients, he meant that if they recovered they did, and if not, they did not. He thought that had been the experience of every one interested in the disease. For the last two years he had been doing something, and his patients had been recovering. He had given the facts and said that Dr. Mills could question the truth of them, but he could demonstrate that they were able to clear up cases not cleared up before.

As to propaganda, that was a criticism which he had had to meet and which was not his fault. The State Board of Charities published a report without consulting him, which was against his wishes. It had injured him, perhaps, he did not know. On the other hand, he thought it was a favorable sign when people began to discuss the question of teeth. When a patient applied at the Mayo Clinic, one of the first things that is done is to look at the teeth. The dentists have the point of view that every tooth should be saved, but he had seen enough of these cases from personal experience to know that is a wrong idea. When people wake up to the fact that a tooth can be examined with the roentgen ray and attended to, the situation will be much better.

He believed thoroughly what Dr. Meyer said. The work must be carried out in spite of remonstrance. The work is accurate, and the fact that the patients recover is sufficient to admit its great usefulness. Their statistics and their work are open for inspection, and any one who has visited the hospital and seen the work has not questioned the value of what they are trying to do. They expect to continue this work and hope to have better reports next time.

GRADUATE NEUROPSYCHIATRIC INSTRUCTION. Dr. Theodore Weisenburg.

All medical centers are concerned with graduate medical education, and efforts are being made in this direction in neuropsychiatry. While each medical center should make every effort to give a first-rate course, nevertheless, all such teaching in this country should be coordinated on a broad, general plan. The Graduate Medical School of the University of Pennsylvania has established courses in neuropsychiatry lasting four months and one and two years, leading to the degrees of Master of Medical Science and Doctor of Medical Science. In the two year course the student may take part of his training in other cities. Experience has shown that courses of less than four months are not of value and should be abandoned by medical schools.

DISCUSSION

Dr. E. W. Taylor, Boston, said that Philadelphia had been a pioneer in inaugurating graduate neurologic courses. Their importance was evident, as Dr. Weisenburg had pointed out. He spoke, however, somewhat slightingly of short courses of a month on this subject and stated, with reason, that it was impossible to "teach neurology" and "make neurologists" in so short a period of time. With this attitude one might entirely agree but this does not invalidate courses of brief duration which naturally are not designed to instruct students fully in any one branch of knowledge but rather to interest them and start them in the right direction. This was all one could attempt to do whether the courses were of one month or from one to two years. Whether a degree is conferred was a matter of secondary consequence. The essential matter is to excite more general interest in the medical profession in this branch of work. He entirely agreed with Dr. Weisenburg that cooperation is advisable and that these graduate courses should be developed in the various medical centers. In the Harvard Graduate School they hoped to establish one and two year courses somewhat similar to that suggested by Dr. Weisenburg, such courses possibly leading to some definite certificate or diploma. Some such plan certainly should be worked out, particularly in the field of neurology and psychiatry, in the attempt to establish more clearly their intimate relationship with other departments of medicine.

Dr. Carl D. Camp, Ann Arbor, Mich., said the Medical School of the University of Michigan did not have a postgraduate medical department but had developed a method of giving postgraduate instruction to certain students who desired to become especially proficient in one particular line of medical work, which should be worth mentioning.

Each year there was selected from the graduating class of the medical school one student by each chief of a clinic (in internal medicine and general surgery, more than one). This man became an intern in the University Hospital for the following year on a rotation service which included various specialties as well as medicine and general surgery. The next year he became a "senior intern" in the clinic of the chief who selected him. During the second year he lived in the hospital and received his room and board, laundry, etc., and a small honorarium. The third year he continued in the same service and received the title of demonstrator. He lived outside of the hospital but received a salary sufficient for maintenance. The fourth year he received the title of instructor and a somewhat larger salary than the previous year. It was recognized that during his third and fourth year, although essentially a student, his services in the hospital in caring for patients, etc., deserved remuneration.

Of course the matter and the kind of instruction differed in the various clinics. It was Dr. Camp's aim in the clinic of neurology to have the senior intern interest himself chiefly in the clinical laboratory investigations, both with reference to routine examinations and research methods. He also carried out certain lines of treatment, such as giving injections, and was urged to develop original methods if possible. The student in his third year was expected to interest himself chiefly in the cases with organic lesions and his research was chiefly in problems of physiology and pathology of the nervous system. In the fourth year the cases with abnormal psychology and social maladjustment were given to him.

Dr. Camp said that each of these men did a certain amount of teaching of undergraduates, chiefly in the details of laboratory investigation and therapeutic measures, and they had the same access to the library of the university as the members of the faculty of the medical school. They had plenty of time for reading and for writing up research problems.

This method solved certain problems, particularly with reference to the development of specialists, which were sources of difficulty in postgraduate schools. The instruction was limited to a selected group of men who showed, while undergraduates, a certain mental adaptability to the special work that they wished to take up. They were guaranteed an intern year in a general hospital where modern methods are used in all branches of medicine. The time was sufficient to enable a good man to become qualified, and still it did not delay unreasonably his entrance to a productive career, and it was equally available to those who had money and those who had none.

Dr. Theodore H. Weisenburg, Philadelphia, said that he had nothing to add except in elaboration of what Dr. Taylor had said in explanation of his reference to a "brush up course." They had been giving similar courses in Philadelphia up to this year, but their experience had been such that they had decided not to continue them, because the students could not gain very much knowledge in a month's time.

The advisability of giving degrees, of course, was a debatable question. The university intended to give degrees to men who passed examinations and were acceptable.

MULTIPLE SCLEROSIS, THE VEGETATIVE NERVOUS SYSTEM AND PSYCHANALYTIC RESEARCH. Dr. Smith Ely Jelliffe.

This paper presents an attack on the multiple sclerosis problem from another angle as conditioned by a vasomotor exudative diathesis with a multiple etiology; an experimental research into the unconscious psychologic material and its possible bearing on faulty compensations in the organic field; unconscious action patterns, over compensatory vasomotor responses causing exudations and consequent blocking or interference with nerve pathways; the relationships to other exudative conditions, such as Quincke's edema, catatonic brain swelling, etc.

DISCUSSION

Dr. Archibald Church, Chicago, stated that he could not sufficiently express his appreciation of the philosophic explication that Dr. Jelliffe had so interestingly set forth, but he confessed disappointment that Dr. Jelliffe had not made some reference to recent investigations as to the spirochetal origin of multiple sclerosis and the observations and experiments that had come forward in the last three years. A spirochete had been discovered in the tissues by at least three independent and separately working observers, and inoculations of the spinal fluid in lower animals had been found to produce conditions identical with multiple sclerosis. This spirochete was somewhat similar to the spirochete of syphilis—a single, spiral form or body which terminated at either end in a hairlike terminal, and had been sufficiently definite to have been independently recognized by several observers.

The investigation of the subconscious in these cases was of academic interest, but he hoped that, with Dr. Jelliffe's broad reach into the literature of all lands, the Association might have some information in regard to this alleged definite etiologic factor, and prospective therapy.

Dr. William A. White, Washington, D. C., said that the remarks of the gentlemen who had previously discussed the paper were the sort of remarks and the reason for which the view propounded by Dr. Jelliffe had been expressed. He had listened with much interest to the paper and was acquainted with Dr. Jelliffe's point of view. He believed that the principles set forth were of immense importance. He also realized that it was exceedingly difficult to have these ideas accepted, as was shown by the discussion of the previous speaker. It was his conviction that the conception as a whole was not a difficult one. It was relatively simple, if it could only be properly expressed.

It seemed to him that it must be conceded that the psyche had a history which was contemporaneous with that of the body, and that the man could not be understood if we limited our conception of him to the period between the time of his birth and the time when he first came under our observation. The individual was connected with his past through the ages by the phylogenetic line of his ancestors and all of those connections must be expressed in him, because the psychologic level of his reactions expresses total reactions.

It seemed to him *a priori* that when there was a serious organic defect in a person, especially one which was phylogenetic in its origin, we must necessarily find, if we know enough to read the signs aright, some explanation of that defect at the symbolic level. That brought up the whole subject of paleopsychology which Dr. Jelliffe had talked about, and the possibility of archaic reactions. If we had an individual whose machinery was archaic, his reactions must be archaic in character. It was only a question of interpretation as to what we believed to be archaic, so that we may be sure that the psychologic symbols were archaic. There might be some question as to whether the symbols that Dr. Jelliffe has set forth were archaic in type, but there could be no question that we had them here, and that their malignancy must depend more or less on their archaic character.

Regarding the infectious origin of multiple sclerosis, he had no doubt that Dr. Jelliffe had that in mind when he spoke of the unity conception—how difficulties of adjustment which grew out of physical imbalances constantly accrued throughout the years until we had certain changes of structure correlated with certain symbols at the psychologic level. Here were organic diseases and there was constant friction, continuous motor sets, which were just the thing that was at the basis of that individual's susceptibility which permitted certain kinds of infection; so that he could see no antagonism between the infectious relation to the multiple sclerosis of spirochete infection and the point of view which Dr. Jelliffe had set forth. We were dealing with the question of energy transformation and energy discharge, and we saw psychic reactions which must be interpreted. We could then learn whether a man had an archaic machine, or whether he had damaged his machine by too great stress. He spoke for unity of the individual. He did not believe it was possible to have any kind of disease, such as multiple sclerosis, without being able to find some correlative symptom at the psychologic level which fitted into the picture.

Dr. Adolf Meyer, Baltimore, supposed the difficulty of the situation arose largely because one could not help creating a suggestion of etiologic potency of the facts which were brought forth and emphasized. He believed, however, that at the present state of knowledge it was best to look on these things mainly as coexistences; as matters concerning which a sense of economy of the individual would largely determine on what phases one wanted to spend one's time in the individual case. The principle of the economy of science forced us to focus our attention on the things actually demonstrated. One had to determine whether one had either part of a vicious circle, or whether there was possibly an actual initiative rôle. Whether that was the case in the conditions as presented by Dr. Jelliffe, he did not know. He did not think that he had made his case from that point of view and he did not know whether he intended to do so.

He was not sure that Dr. White was not rather maintaining the general doctrine of unity of the organism than any special conviction that we ought to assume that the presentation of Dr. Jelliffe had actually demonstrated that psychogenic factors were potent in multiple sclerosis. He happened to come from one of the centers of multiple sclerosis production. He did not think that it was best at present to say that multiple sclerosis selected persons with archaic type of mind or with anal erotic tendencies, many of which were iniquitous. There is the problem, which had been discussed, concerning the infection of teeth. It was essential that we should be pluralistic, and yet should allow the sense of economy of scientific thought to be rather intent on the question: Does the matter under discussion merely form an incident or possibly part of a vicious circle, or does it have an actual etiologic significance?

Dr. Smith Ely Jelliffe, in closing, said that after Dr. Church had given him credit for erudition, he could ill repay this compliment by confessing ignorance of the whole mass of recent literature concerning the finding of a new spirochete in some patients with multiple sclerosis. Of course, that was an old story. He thought that Dr. Church had probably seen for the last two years in the *Journal of Nervous and Mental Diseases* his abstracts of this work, not only of Kuhn and Steiner's original claims but of Siemerling and others, and vigorous protests concerning the whole subject. In fact, in the May issue of the journal a critical collective abstract and careful discussion of the whole of Kuhn and Steiner's theory was to be found. He thought he had made it perfectly clear that this type of case was outside of this discussion; such types, or any new ones, could be included under the specific infectious types of disseminated cerebrospinal disease. In reply to Dr. Church, he would be discourteous not to acknowledge the validity of his point, but he had already excluded from his discussion that whole group. He did not know how many multiple scleroses might be left after the exclusion of all similar types of infection. He was not sure that we would have any multiple sclerosis left if we could go sufficiently far into all possible secondary factors. That he did not know, nor could learn from any one else, but he had assumed, on the basis of pathologic findings, that a special group characterized by features belonging to the exudative diatheses was possible. This was the group that interested him in this communication to which he added the findings he had reported.

In answer to Dr. Meyer, he was a thorough believer in the economy of the situation. Naturally, it would be more or less incidental, because should he ask the gentlemen here how many cases they had treated, there would be one, two, six, a dozen or more, excluding records of polyclinic material, so that from the standpoint of time and labor, his contribution to the problem of multiple sclerosis would be more or less incidental. That was not the point in particular, but the whole problem of the exudative diatheses might be entered into. He might enlarge the conception and include the whole group. In that case we might attack catatonic brain swelling, certain arthritides, certain skin exudative phenomena, etc., and many other conditions from a similar point of view. We find in the realm of general medicine groups which have been classified under the exudative diatheses, to the study of which the principles here brought forward might be applied. Here we enter into many problems of physical chemistry, and to his mind a great deal of value might be thrown on these problems by adequate exploration of the unconscious. But to confine his remarks to the present communication, all he contended was that in certain cases of certain conditions called multiple sclerosis, certain things were found in these patients' unconscious. For those who were interested in the probing of the unconscious, deep sea fishing we may call it, it would be interesting and might be important, to learn from others the results of their "soul plankton" findings.

ABDOMINAL NEURASTHENIA. Dr. Alfred S. Taylor. This article will appear in an early issue.

DISCUSSION

Dr. Walter Timme, New York, said that the cases cited by Dr. Taylor were of extreme interest, especially to those who had done work on the abdominal sympathetic. They bore out very well the surgical part of the picture which neurologists had looked for. Some years ago he had done some experimental work on animals along this line. This work was divided into two groups; in one he diminished the vagus activity by ligating the abdominal vagus just before it entered the diaphragm; in the other series he ligated a great many branches of the sympathetic. By these means he produced artificially, respectively, sympathicotonic and vagotonic states. He obtained interesting results in the animals; these results resembled those seen by Dr. Taylor in man. In the animals in which the sympathetic filaments were tied off, the work was done so that only part of the conduction was interfered with. The abdomen was then closed and the animals allowed to live three or four months. In those cases in which the vagus was allowed to be free a condition of general ptosis was found in the abdominal organs, a high degree of motility, lack of tone, a lack of rotundity of the abdominal muscles and malposition of all of them. The animals were killed one and a half hours after the ingestion of food, and the stomach was found practically empty. In those cases in which the vagus was tied off, the stomach and the large colon were increased in size, and their walls hypertrophied. With this dilatation and hypertrophy there were all manner of kinks in various portions of the ascending colon and the sigmoid, with pronounced enlargement. In those animals there had been retention, that is, six hours after ingestion of a full meal the stomach contents were still in the stomach, there was very little motility, and conditions were obtained much like those Dr. Taylor found in man.

With these disturbances in the vagus and sympathetic in man, there are certain vasomotor and blood pressure alterations which are allied to fatigability, low carbon dioxid tension and blood sugar disturbances; and there is a condition which has been dubbed by many as "abdominal neurasthenia."

The condition which was produced artificially by ligating is frequently caused by various procedures, that is, any torsion of the diaphragm in malposition may cause torsion and pinching of the vagus, and may produce the same symptoms that were brought about artificially. Second, toxins affecting the suprarenal glands may do almost as much toward blocking the sympathetic, and there results a train of symptoms referable to the suprarenals plus the gastro-enterologic findings.

If that condition is seen early enough, that is, before the adhesions due to ptoses are formed, a great deal can be done therapeutically. After the adhesions are formed, we must proceed surgically, and relieve the conditions produced by the kinks and adhesive bands. If it is realized that these conditions of pain, hyperacidity, vomiting, loss of weight, general depression and fatigability may be produced by such an actual terminal local condition as that described by Dr. Taylor, we are in a fair way toward relieving many of the patients that create a great deal of disturbance in our life. They are referred to us usually with a letter something like this: "The patient whom I am sending you with this note is a young lady who for the past four years has had vomiting with attacks of pain. She has had three operations. We removed her appendix and her gallbladder, and then we took out her ovaries and uterus, and now the poor girl has nothing left—not even hope. Please help us out." These cases are becoming more frequent, but because many surgeons recognize the true situation, we now frequently see them before operation is necessary.

Dr. Timme thought that Dr. Taylor's work should be noted and that further work in this field would be productive of much good.

Dr. Smith Ely Jelliffe, New York, said that Dr. Timme's narration of his patient reminded him of approximately 50 per cent. of his own. They came to him with no ovaries, no teeth, no intestines, no hope, no pocketbook, no anything—and then he was expected to put them on their feet. He did not always do it, naturally; but, working along the lines that Dr. Timme had already indicated, that is, with the concepts of the physiology of the vegetative nervous system and with the belief, which he did not mention but which he knew he sustained, that emotions may act exactly like toxins, he had cured some of the people who did not have anything left inside of them after the surgeons had tried to cut disease out of the body.

Dr. Alfred S. Taylor, in closing, said the essential point of the paper was that these patients had been suffering from the effects of organic anomalies that would prevent therapeutic treatment, in the way of internal medicine, from working satisfactorily. There was a mechanical blocking of partial degree in every one of them, obvious in the roentgen-ray examination and confirmed on the operating table. The correction of these mechanical difficulties resulted, without other treatment, in an entire clearing up of the various nervous disorders, together with the digestive disturbances.

He knew it was unpopular to refer to intestinal toxemia and similar states of the intestinal tract, and appreciated how many ills had been laid at its door without good reason. However, if there were any blocking, even if not complete, if there were slight disturbances of the digestive processes every minute of the twenty-four hours, it was easy to believe that they influenced the nervous system deleteriously, and that if corrected, the disturbances, both of digestion and of the nervous system, would disappear.

The only conclusion he would like to draw from the presentation of these cases was not that one can cure every case of neurasthenia with these procedures, but that in treating a case of neurasthenia with abdominal symptoms it was only fair to the patient to make a roentgen-ray examination of his digestive tract to find out wheher there was something organic that might be corrected, and then to be guided by the findings.

THE SIGNIFICANCE OF PAIN IN THE FACE IN DETERMINING THE LOCATION. Dr. Williams B. Cadwalader. This article appeared in the August issue, page 182.

DISCUSSION

Dr. Malcolm A. Bliss, St. Louis, said that it might be of interest in this type of case to recite one that occurred recently in his experience. A woman about 50 years old was sent to him for a fifth nerve disorder—for tic. The striking fact was that pain in the face extended over the distribution of the first and second division. She kept her jaws rather tightly closed, apparently because it gave her intense pain to open them. On investigation it was found that by digging deep behind the ramus of the jaw on that side there could be felt a mass that had extended across the base of the skull in such a way as to involve, outside of the skull, the first and second divisions of the fifth nerve.

Dr. Harvey Cushing, Boston, said that he could subscribe fully to all that Dr. Cadwalader had said. He had had a series of forty-five verified acoustic tumors, and he thought that the chronology of the symptoms was the most important means of making a diagnosis. Sometimes the patients are not intelligent enough possibly to appreciate the exact order of the onset of the symptoms, but in nine out of ten cases, on questioning them, they will recall that the tinnitus and the oncoming deafness preceded the symptoms that point to cerebellopontile involvement or those that indicate general evidence of headache and choked disk. Sometimes the interval is very long. He had now a young man in the hospital who, seventeen years ago, had a sudden attack of deafness and then no further symptoms until he began to have headaches, choked disk and evidences of cerebellar involvement. He proved to have an acoustic tumor.

The trigeminal tumors are much more rare, and they begin in the way Dr. Cadwalader mentioned—with pain in the face—and they tend, in their slow growth, to spread posteriorly and to get into the posterior fossa, so that deafness, and possibly facial paralysis, may come at any time. They, too, are slow growing lesions, and although it is a difficult procedure, the sensory root division in these cases gives a great degree of relief, and the patients may go on in comfort for a number of years. Thus, a long period of symptomatic relief may be assured, although the procedure is purely symptomatic and palliative.

Dr. Williams B. Cadwalader, in closing, said he was in accord with all that Dr. Cushing had said, but wished to emphasize the importance of ascertaining the exact order in which these symptoms developed. If pain begins early, it may indicate that the tumor is growing in the region of the gasserian ganglion. On the other hand, if deafness begins first it may mean an acoustic tumor growing in the cerebellopontile angle.

SECTION TOTALE DE LA MOELLE DORSALE PAR CONTUSION RACHIDIENNE AVEC CONSERVATION DES REFLEXES ROTULIEN ET ACHILLEEN GAUCHES ET PRESENCE DE REFLEXES D'AUTOMATISME MEDULLAIRE (COMPLETE SECTION OF THE DORSAL SPINAL CORD THROUGH SPINAL CONTUSION WITH PRESERVATION OF THE LEFT PATELLAR AND ACHILLES' REFLEXES AND THE PRESENCE OF REFLEXES OF MEDULLARY AUTOMATISM). DR. GUSTAVE ROUSSY, Paris, France (by invitation).

The patient suffered from total interruption of the spinal cord as a result of a contusion. He was observed by Dr. Roussy eighteen months after this injury. At this time the clinical examination revealed the following condition: There was a total flaccid paraplegia with loss of both superficial and deep sensation up to the tenth thoracic segment; there was conservation of the left knee jerk occasionally accompanied by knee clonus, likewise conservation of the left tendo Achillis jerk; in addition, there were present the so-called reflexes of medullary automatism which could be induced on either side by irritation of the cutaneous surface by pressure exerted on the malleolar region and likewise by the so-called Pierre Marie-Foix procedure. A fact of importance in this case was that some of the reflexes of medullary automatism could be provoked likewise by injecting into the cystostomy opening fluids for irrigating the bladder, a bilateral mass reflex occurring not only when the fluid was introduced, but also when there was any change in its temperature or pressure. Finally, there was observed in this case, aside from the sphincter, genital, vasomotor and pilomotor disturbances, which were marked, a most pronounced hyperhidrosis.

Anatomically, there was complete disappearance of all medullary tissue on a length of 1.5 cm.

Histologically, it was proved beyond doubt by all staining methods, and particularly by the Bielschowsky method, that there was no medullary substance left at the level of the lesion. In the meninges, located on the anterior surface, a number of root bundles were discovered that contained regenerating fibers.

The hypothesis was advanced by Dr. Roussy that it is more logical to suppose that the inferior segment has progressively recovered its function of medullary automatism, which became manifest through the reappearance of some of the tendon reflexes, as well as by the presence of the so-called reflexes of defense. The lapse of time has to be remembered, the fact that the patient was observed, as was said before, eighteen months after the initial lesion. In terminating, the point was emphasized that the observations recorded during the war served to approximate more closely the results of clinical investigation and the facts already demonstrated in the domain of experimental physiology, and that we can in a certain measure speak of spinal man in the same way that we speak of spinal dog.

DISCUSSION

DR. PETER BASSOE, Chicago, asked Dr. Roussy in regard to the plantar reflex in this case, if its condition would possibly have some bearing on the problem of the nature of the Babinski sign.

If he was not mistaken it had been found that in total section, with subsequent return of the tendon reflexes, the Babinski sign has not been obtained. He was told by men who were in the war zone that its appearance after injury of the cords with paraplegia was looked on with favor as indicating that a total lesion did not exist. If a Babinski sign should be obtained in a case of proved total section, it would show that no cerebral component is necessary, and that the extensor response may only be part of a mass reflex.

Dr. Williams B. Cadwalader, Philadelphia, said that the subject that Dr. Roussy had brought up was very interesting, particularly after some of the experiences in France during the war.

Many will remember the report of a case, in 1901, by Dr. Stewart of Philadelphia in which there had been a gunshot wound with complete section of the spinal cord at the level of the tenth dorsal segment. Operation was performed by Dr. Stewart and the spinal cord was reunited by sutures. Subsequently it was claimed that return of function had occurred. Dr. Weir Mitchell expressed his skepticism as to this result at a meeting of this Association held at that time. He had known the patient well and had observed her more or less during these years; had examined her carefully, and had just prepared a report (published in the *Annals of Surgery*, June, 1920) of her present condition. There can be no doubt, so far as the clinical signs are concerned, that she still has complete division of the spinal cord with no return of function. The case presents all the automatic reflex movements which have just been so carefully described by Dr. Roussy. The picture presented is like that described by Head and Riddoch of England, but is slightly modified. The limbs are spastic; there is a permanent Babinski sign—in fact, a typical "mass reflex"—and no return of function, in spite of the fact that nineteen years have elapsed since the spinal cord was reunited.

Dr. Harvey Cushing, Boston, said that many years ago—in 1900, he believed—he had a patient with a traumatic total transverse lesion of the cord, as was supposed. He was well taken care of and had a return of all the deep reflexes. The case was thoroughly studied and innumerable sections made of all the spinal segments. As far as could be found, there were no fibers passing through the seat of the lesion. He had expected to find some fibers because he had been imbued with Bastian's theory of complete flaccid paralysis after total transverse lesions.

He had had the opportunity of seeing such cases with Drs. Head and River, and the extraordinary mass reflexes they have described are interesting. They, of course, account for the cases that have been spoken of, in which there has been a return of function after suture of the cord. The one thing he wanted particularly to mention was his surprise that in this patient of Dr. Roussy's so long an interval had elapsed before the reflexes returned, because in Head's cases these automatic reflexes began to return quite early, sometimes in two or three weeks. As he understood it, too, it is because in these cases there have been no bed sores or infection, and he believed that it is only in the absence of infection that the cells of the distal portion of the cord succeed in regaining their function. If bed sores, cystitis and renal infection occur, the resultant toxins appear to prevent the cells from regaining their function, so that a total transverse lesion may continue to have the picture we formerly associated with it.

In this early study which he made twenty years ago, he felt a few fibers might possibly have been overlooked in the scar. He asked Dr. Head about this, and he admitted that a few fibers might possibly have been preserved in the cases he was studying. They are difficult to detect even on careful postmortem histologic studies.

However, some four or five years ago he treated a poor woman with a metastatic carcinoma in the lower thoracic spine, symptoms of which appeared some twenty years after amputation of the breast—one of the late cases. She suffered greatly from pain and spasmodic contraction of the legs. She continued in fairly good health, though praying for death as a release from her suffering. He should possibly have made a partial division of the tracts in the cord, as suggested by Drs. Spiller and Frazier, but instead he deliberately divided the cord completely four or five segments above the situation of the lesion. It is a simple and dry procedure. This woman, who had been taken care of in the hospital, was exposed to none of the accidents of the soldier, who had to be transported and was likely to get bed sores and infection. Here, then, was a case of unquestioned complete surgical transsection of the cord. The patient had an early return of these mass reflexes, like those Dr. Roussy so clearly told about. She lived in great comfort for several months, free from pain.

Dr. Hugh T. Patrick, Chicago, asked Dr. Roussy how he explained the return of the knee jerk and Achilles' jerk on the left side and not on the right; unless, of course, the lesion was lower on the right side, which he had not understood to be the case.

Dr. Gustave Roussy, in closing, replied to Dr. Bassoe that at no time was the Babinski phenomenon, or its normal plantar reflex, demonstrable.

Regarding the point brought up by Dr. Cushing as to the period of time which elapsed before the return of the tendon reflexes, nothing definite could be stated as the patient did not come under his observation until eighteen months after the injury, and only fragmentary information was obtained concerning his condition prior to that time.

In answer to Dr. Patrick, no plausible explanation could be given for the fact that the tendon reflexes reappeared on the left side only. The reflexes of defense, however, occurred on both sides.

Book Review

MANUAL OF PSYCHIATRY. By Aaron J. Rosanoff, M.D. Fifth Edition. Pp. 624. New York: John Wiley & Sons, Inc., 1920.

Psychiatry moves. Editions are milestones. In the first edition (1905) of this manual, the practice of psychiatry was dealt with in one chapter: it now fills ten. New descriptions of mental measurements appear, psychanalysis, community surveys, social service, after-care.

The de Fursac translation, much revised, is still the center of Parts I and II, but there are many explanations and interpolations by Rosanoff, Hollingworth, Neymann and Miss Jarrett. There is a tendency to turn from the general to the particular. The result is a book, patchy but clear, in which the reader can easily find his way. Part III (200 pages) contains detailed instructions for performing intelligence tests, examinations of spinal fluid and special diagnostic procedures which will gratify the physician who needs a very definite help in an unfamiliar field.

The psychiatrist will wonder at the dispassionate expositions of psychoses and treatments from irreconcilable points of view. The older psychotherapy is on one page and psychanalysis on another. But why not?

Fifty cents each will be paid for the April and May, 1919, issues of the Archives of Neurology and Psychiatry. Address to American Medical Association, 535 North Dearborn St., Chicago, Ill.

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THE SPECIAL FIELD OF NEUROLOGICAL SURGERY AFTER ANOTHER INTERVAL*

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Those engaged in scientific or professional pursuits have need of a periodical accounting of stock no less than those engaged in business. The results of such an inventory may sometimes be of more than personal interest, especially when projects have been undertaken which are in a measure novel or special, for their success or failure may encourage others or may deter them from similar adventures.

SPECIALIZATION IN GENERAL

In a sense, I suppose, every medical graduate tends in time to particularize to a certain extent, and through liking or opportunity comes to be known as more expert or better informed than his fellows in one thing or another. He may have had an unusual experience in relation to child-birth, shown unusual skill in dealing with the ailments of children or with the maladies to which their mothers are heir. He may have a mechanical bent and be particularly apt as a setter of broken bones or a corrector of deformities. He may be a particularly good microscopist or show greater facility than his contemporaries in the use of some other precise instrument of importance in diagnosis. He may have become engrossed in some special disease, indeed of some particular organ, or what is more, of some special form of treatment for a particular disease of a particular organ. His interests may become purely scientific or purely sociological and take him away from the bedside entirely. Of all this there is no end or ever will be. For there are ways innumerable in science or practice in which we as individuals or groups of individuals come to particularize in our work.

The participant in this subtle process of specialization may not always be conscious of it, or, if he is conscious of it, may not wish to admit the fact even to himself, and would encourage his employers, be they trustees, patients or the commonwealth, to believe that he is equally capable of dealing with all branches of his larger subject. On the other hand, the concentration of effort may be intentional and represent a desire to contribute to knowledge, rather than income, though they are not necessarily divorced, and for this the individual must perfect himself in certain directions rather than others.

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* This address was made to do double duty before the Tri-State District Medical Society at Waterloo, Iowa, Oct. 7, 1920, and the Cleveland Academy of Medicine, Oct. 8, 1920.

Thus the specialist and specialties arise, but in the case of each of them, should progress cease after a generation or two, it is inevitable that the imitators of the pioneers will diminish in number as well as quality. And when this time comes, following a period of more or less disrepute, the particular species of specialist dies out, and the prodigal specialty returns to, or should return to, its parents. If this happens, the reunited household will unquestionably have profited by the episode.

Medicine has grown in the fashion of a banian tree. In the beginning there was a single stem. This, in the remote past, each professional aspirant learned to climb, and in his lifetime could encompass without difficulty all the knowledge its various branches represented. But from the main branches which the original tree put out, particularly from physic and surgery representing the application of medical knowledge to practice, there soon dangled many roots. Some of these finally reaching the ground became attached, and drawing up their own nourishment have in some cases enlarged and become permanent and necessary supports to the parent branches.

Modern aspirants sometimes climb these outlying props, mistaking them for the main stem or believing that they have found a short and less crowded way to the top where others they wish to emulate are seen to perch. The temptation is great and to some irresistible, but short cuts to specialization without thorough preparation in the fundamentals make incompetent if not dangerous practitioners, and so long as this is permitted within the profession itself we should be less intolerant of those who have smuggled themselves into the tree with no medical preparation whatsoever—the osteopath perched insecurely on a limb propped by massage and the Christian scientist tip-toe on another feebly supported by psychotherapy.

Be this as it may, in time there has come to be a veritable grove of trunks, and every season new roots become grounded in such increasing numbers that it is difficult for the untutored to realize that the growth really represents a single tree. To be sure, an outlying prop to a limb may occasionally dwindle and disappear as botany has disappeared: or, after a period of temporary vigor, it may become incorporated with an adjacent and more vigorous one, but the general tendency is for a further and rapid multiplication rather than for any reduction in number, for, though no longer needed, an old prop dies reluctantly if once it has come to support an important branch.

Near the center of the tree are two particularly large parent branches, one of them perhaps more flourishing and well supported of late than the other, but both of them apparently essential to the welfare of the tree. From legend alone do we learn which of the two probably represents the main stem and which the secondary offshoot. Whether physic was a specialty which branched off from general Medicine before surgery, or surgery before physic, is not quite clear.

In its westerly transplantation to different countries in successive eras the tree of Medicine, to be sure, has not always put out the same branches in the same way. We may recall the words of the distinguished Philadelphian, John Morgan, who in 1765, on his return from Edinburgh, announced in his justly celebrated "Discourse" his intention of limiting himself henceforth to "those cases which belong most immediately to the office of a Physician" and of no longer "interfering in the proper business of Surgery." From this it could be argued that the practice of physic thus withdrawn from general Medicine, which included handicraft, became in this country at least the first avowed specialty. For the real standard of specialization is the determination to avoid diffuseness by withdrawing from participation in the wider field and by devoting oneself to a more limited one.

One might pursue this subject further and trace the development of some of the various secondary offshoots both of physic and surgery in the past and see what has happened to them—whether they have taken secure root and continued to flourish or whether after a generation or two they have either died back or become incorporated again with one of the main stems by fusion—but interesting as this might be, it is not the purpose of this essay.

New specialties at present undreamt of will continue to arise. It takes little to start one. The diseases incidental to a novel occupation like aviation; the introduction into the clinic of a new discovery like Wassermann's reaction, or Roentgen's ray; a new instrument of precision like the calorimeter—such things as these will make a succession for all time of unlooked for opportunities for us and our followers in Medicine to engage in new endeavors under novel conditions.

These must be regarded as accidental occasions for specialization, to be distinguished perhaps from the purposeful determination to specialize within an established branch long occupied. But under both circumstances those who can best take advantage of existing opportunities, or can originate others, not only must have had a good general training in clinical medicine and surgery, but must have been thoroughly schooled in the fundamental subjects—in the anatomy, physiology and chemistry of morbid as well as of normal tissues and organs—for without this knowledge any special branch is supported by a root lodged in sand which does not long survive overloading. There is only one way to get a secure seat on any outlying branch and that is by approaching it from the main stem, no matter how wearisome, laborious and time-consuming this process may be.

SPECIALIZATION IN PARTICULAR

In 1905[1] and again in 1910[2] a paper was read before the Cleveland Academy of Medicine on this selfsame subject of Neurological Surgery as a specialty. The first of them was written shortly after the decision was made to limit my operative surgical work to what seemed to my several advisers, and indeed to me at the time, to offer small pickings. There had dangled for a long time from the branch of surgery, if we may continue with our banian-tree simile, a wisp of root over a very unpromising patch of soil. It had become attached on more than one occasion, though feebly to be sure, and had been tested thoroughly by Agnew in this country and later by von Bergmann in Germany; it had for reasons not far to seek been abandoned as both unfruitful and unsafe. Later on, Victor Horsley turned his brilliant mind to the subject with far greater success than his predecessors, though he showed more interest and perhaps skill in its physiologic than in its purely surgical aspects.[3] Specialization is an uncompromising mistress. It was an avocation of Horsley's, not a subject to which he limited himself, and though he had imitators he left no disciples.

It hardly seemed possible fifteen years ago that the surgery of the nervous system by itself could furnish material enough to occupy a surgeon's undivided attention and insure him a livelihood, far less that it would promise opportunities within itself for further specialization on subjects like tumors of the brain in general or tumors of the pituitary body in particular. Time has shown, indeed, that these early misgivings were unfounded, and that there is not only an appeal but abundant opportunity for special workers who plan to restrict themselves to this outlying branch is evidenced by the fact that a number of surgeons interested in the nervous system have organized themselves into an Interurban Neurosurgical Society in the expectation thereby of making more rapid progress in this specialty through an intimate interchange of opinions made possible by clinical meetings.

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[1] The Special Field of Neurological Surgery, Cleveland M. J. **4**:1 (Jan.) 1905; Johns Hopkins Hosp. Bull. **16**:77 (March) 1905.

[2] The Special Field of Neurological Surgery: Five Years Later, Cleveland M. J. **9**:827 (Nov.) 1910; Johns Hopkins Hosp. Bull. **21**:325 (Nov.) 1910.

[3] One of his colleagues, indeed, at the National Hospital went so far as to make a study of the brain tumor cases operated on there, and concluded that the average duration of life of patients thus afflicted was longer without than with an operation.

It is impossible within the limits of an address to enter at all fully into an account of all that has transpired in the field of neurologic surgery during the past decade, far less to give anything more than hints of what lies before us in the way of problems. I shall restrict myself, largely, to some of the topics which have engrossed the attention of my co-workers and myself during the interval, though they represent but a small portion of the larger work in which many have become engaged.

To conform more or less to the plan of my former papers I shall allude in turn to the surgery of the brain, the cord and the peripheral (cranial and spinal) nerves,[4] with such comment and digression as I may be led into under these separate headings. But before entering on these subjects I desire to say something about a mysterious personal quality necessarily possessed in varying degrees of perfection by every one who does something with his hands—namely, his technic.

NEUROSURGICAL TECHNIC

Perfection in the conduct of his therapeutic measures is as essential for a surgeon as is the technic of laying on colors for an artist or of producing sound for a musician. One may have abundant knowledge of art or music or medicine and yet be a poor performer. In surgery a proper technic is by no means the only element of success, but it is an important one.

Let us look back for a moment on the conditions at the beginning of the century, when from our present standards the methods of conducting both cranial and spinal operations were crude beyond the belief of our juniors who happily know better times. The day of the mallet and chisel for opening the skull was not far removed. An exploratory exposure of the brain was looked on by the prospective patient with the same dread with which an abdominal operation had been regarded a generation before, and by the surgeon with comparable misgivings. Few, if any, operators ventured to undertake one of these procedures except under the direction of a neurologist, and only then when conditions had advanced to such a point that the diagnosis could be made beyond peradventure, though by this time, alas, in the presence of tumor, tension within the skull had become extreme. This made a procedure with inherent technical difficulties extremely hazardous, not alone at the moment of its performance but the ensuing complications due to imperfect and hasty closures led with disheartening frequency to complications which sometimes prolonged an existence worse than the original disease.

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[4] There is, too, a surgery of the sympathetic nervous system, poorly developed it is true and hardly recognizable even in this its experimental stage. We have had glimpses of it in certain operations for goiter, in certain forms of treatment suggested for Reynaud's disease; and the proposals by Lèriche, in regard to a purposeful surgical paralysis of the vasoconstrictor nerves which accompany the blood vessels, are straws to show the direction of the wind.

The influence of the vagus and the splanchnic nerves on the viscera; the relation of emotional states to metabolic stability; Cannon's experimental anastomoses between phrenic and cervical sympathetic, with their remarkable sequelae—these things indicate what may lie before us if imagination is turned to the application of some of the facts known to physiologists regarding the autonomic nervous system.

The most crying need, therefore, in the early years of the century was for an improvement of our technical methods under the realization that what had sufficed for other organs and tissues, when applied to the surgery of the nervous system, was disastrous. Therein lay the failure of Agnew and von Bergmann.

Fifteen years ago we were scarcely at the dawn of an understanding of purposeful decompression for brain tumors. To be sure, the observation had been made and reported more than once that after an occasional osteoplastic exploration which either revealed no growth, or one supposedly unremovable, an entirely unexpected amelioration of subjective symptoms had taken place. However, inasmuch as most of these early operations had been confined to the more accessible central region of the hemisphere, these subjective benefits were more than offset by postoperative paralyses from protrusion of the tense brain through the cranial defect. When the idea of a purposeful decompression over relatively "silent" portions of the brain, conducted, moreover, in areas like the temporal and suboccipital regions where an undue extent of protrusion is checked by securely closed extracranial muscles—when this idea finally took hold, the old explorations which had resulted in a certain measure of relief were resurrected and tabulated as decompressions in our modern sense, which they were not either in reality or intent.

With the development of a reasonably safe and satisfactory procedure for relieving tension both for cerebral and cerebellar lesions, and one which of itself did not lead to paralyses, these palliative operations began to multiply and it soon became apparent to all that the accepted views regarding the cause of what was called optic neuritis, resulting in atrophy and blindness in tumor cases, would have to be revised. For it was obvious that an inflammation of the nerve could not explain a process capable of being checked by the mere relief of pressure, so long as the tumor, to which the supposedly toxic neuritis had commonly been ascribed, remained unremoved.

Inflammations of the optic nerves due to toxic substances and capable of producing atrophy doubtless exist, but changes in the nerve head observable to the ophthalmoscope are infinitely more often due either to an increasse of cerebrospinal fluid tension or to direct pressure on the nerve by tumors in the region of the chiasm than to any other cause—so much more often that rhinologists and ophthalmologists must pause before venturing to make the popular diagnoses of the day which lead so often to unnecessary operations on the accessory sinuses.

I cannot speak for others, but for myself the subtemporal and the suboccipital operations are, in the long run, the two most useful procedures in craniocerebral surgery though, as is true of all operations, they are by no means perfected, and there are right and wrong ways of performing them.[5]

The *subtemporal decompression* I would place first on the list. It may be employed as a temporizing measure even in the presence of a localizable lesion, but it is of chief value in all unlocalizable cerebral tumors. Naturally, since the bone defect is purposefully made over the relatively silent temporal lobe, the growth may at times be unexpectedly disclosed. In the presence, moreover, of a questionable cerebellar lesion, the existing symptoms being hardly such as to justify the more elaborate and difficult posterior exposure, the measure may be used as an aid to diagnosis. For the existence of an obstructive hydrocephalus can be determined under these circumstances by a puncture of the temporal horn of the lateral ventricle, a matter of considerable localizing value, though it is to be admitted that in the presence of a hydrocephalus a decompression over the cerebrum gives but slight pressure relief.

The routine operation which may be regarded as of second importance is the *combined osteoplastic exploration and decompression.* With the boneflap so placed that its base is in the temporal region, the squamous wing of the temporal bone may be rongeured away after the flap is reflected. Though surgeons in the past have sacrificed without hesitation large portions of the calvarium in eradicating tumors, and occasionally this may be necessary even now, yet it is highly desirable that the cranial chamber should be kept as intact as one's skill and the conditions permit. It is also desirable for many reasons that an operation for tumor should be conducted whenever possible in a single session, and the old two-stage performances, provided there is careful blood-stilling, are less and less frequently called for.[6]

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[5] I would have felt ere this that the principles of a subtemporal decompression were so well understood that it was a safe measure in the hands of every general surgeon, but we still see so many sorry results of so-called decompression operations with bone flaps elevated in the region of the temporal attachment and often on the left side, that the happy time does not seem to have come as yet. A detailed account of the measure, long delayed in publication, has just appeared in the "Textbook of Surgical Diagnosis and Treatment," by American authors, edited by A. J. Ochsner **1**:407, 1920.

The third procedure, the *typical cerebellar exposure,* is a still more difficult operation and necessitates elaborate preparations and skilful team work if a long series of these measures are to be carried through with a minimal mortality. In this performance the steps are practically the same, whether it resolves itself into a decompression or in the more or less complete removal of a tumor, should one be disclosed. It is a long two-hour operation at best—an hour for the full exposure and another for wound closure—but when tension is great, when there is threatened respiratory difficulty so that a ventricular puncture in the course of the early stages is called for, or when there is a recess tumor which requires long and careful manipulations, or a tumor of the mid line which necessitates removal of the arch of the atlas and prolongation of the dural incision down over the spinal canal as far as the axis, the performance may well require an extra hour or two.[7]

Since the war, influenced by DeMartel's experiences as well as by our own, I have made a few of these cerebellar exposures under a local anesthetic, and though it is possible, I do not feel that I can perform quite as perfect an operation in this way as when the patient is etherized, even though this in itself adds an element of danger. Under a local anesthetic the long seance becomes still more protracted, and by the time the patient begins to ask if you are not nearly through one can hardly resist the temptation to exceed his established speed limit, to cut corners and thereby run the risk of accidents.

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[6] When a tumor extirpation is anticipated, as in the case of a large endothelioma, a two-stage operation admittedly continues often to be required for there is apt to be undue loss of blood during the elevation of the flap, and there is almost certain to be still more in a certain stage of dislodging the tumor. Under these circumstances I feel that a dural opening at the first session should be limited to the temporal region to allow a protrusion in correspondence with the subtemporal defect—in other words, with the area of bone rongeured away from under the temporal muscle. This will permit the upper portion of the flap to be replaced over an intact dura to await a second session, a certain measure of pressure relief being meanwhile secured. Should the dura be widely reflected at the first session and the tumor exposed with the inevitable protrusion of the hemisphere, this would make one of two things necessary—either an immediate attempt at tumor extirpation, which might be more than the patient could stand, or stripping off the reflected area of bone and replacement of the scalp alone, a procedure which leaves an unnecessarily large cranial defect. One may be compelled occasionally by poor judgment or force of circumstances, to accept one or another of these awkward situations.

[7] A neurologist who recently had the endurance to see an acoustic tumor operation on one of his patients carried through to its conclusion with detailed wound closure, layer by layer, suddenly exclaimed: "I see now why my patients who have recovered heretofore after cerebellar operations have all had bulging necks. I had come to think it inevitable."

These, then, are the three standard types of operation, though there are innumerable modifications of them. Each surgeon in accordance with his particular training and tastes will have devices of various kinds to overcome difficulties as they arise. These technical idiosyncrasies do not affect the three main issues—the exposure of the lesion, its safe and proper handling, the painstaking closure necessary for a wound which may subsequently be subjected to considerable tension.

I have emphasized before, and must emphasize again, that there is no field of surgery in which fastidiousness is more essential to success. Imperfections in technic in the course of operations on other parts of the body, where other tissues than those of the central nervous system are concerned, may delay recovery but not necessarily impair an end result. But corresponding slips which compromise the function of the nervous system may sometimes leave mental or other disturbances which are irrecoverable and to which death is preferable.

The surgeon who feels a due sense of responsibility regarding these difficult cases and who would reduce postoperative deformities and accidents to the minimum must often put all the reserve he has into a single operation. Without injustice to the patients more than one stereotyped measure, like a trigeminal root avulsion or subtemporal decompression, may be undertaken in a single morning, but in the case of the more arduous and uncertain procedures the physical strain and responsibility may be such that he would be foolhardy and negligent of his patients' welfare who would venture on a succession of these tasks.

Then, too, there is no group of patients who require a more detailed preoperative study, in which I feel that the surgeon is obligated to engage, and this with attention to laboratory studies which, in a new specialty, are particularly to be encouraged, is about all that can be properly attended to. It is not the number of cases operated on which have made contributions in surgery possible, but the detailed and careful analysis of the few, in conjunction with allied laboratory investigations.

Surgical technic may seem to be a thing entirely apart from the bedside study of patients preliminary to operation and from coincidental investigations in the laboratory.[8] In a certain sense it is a thing apart, and we are all familiar with men whose operative technic exceeds their judgment as to when it should be put to use. We have all known, too, surgeons who were somewhat awkward craftsmen but who possessed such a thorough knowledge of disease and its processes as to atone completely for their possible lack of manual dexterity. Of the two, the latter are often more safe. Surgeons with both qualities highly developed are, alas! not a common breed. The patient, unfortunately, though he pays admission, has not the privilege of viewing the performance, else he might like to see his appendix removed with a flourish, and to applause. His active interest begins when the curtain falls and he is then apt to find himself more comfortable if the audience thought it a tedious and dull show.

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[8] Though there may be differences of opinion in the matter, I personally feel that for the development of surgical technic no place is comparable to the experimental laboratory, and I feel that every young surgeon should begin to acquire his operative training in a series of operations on the lower animals. Certainly in the case of neurologic operations one should learn on the

In the manufacturing arts, particularly in this country of ours, subdivision of labor has developed to such an extent that no workman makes a completed object. He may excel in the rapidity with which he may drive rivets, in the skill with which he may attach a given piece to an article, of whose remaining parts he has little knowledge

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lower animals the art of trephining, of the use of bone wax, of handling the cord and brain without the production of contusions and extravasations, of dealing with the cerebrospinal fluid which is the keynote of many intracranial operations. The most clever, neat and skilful of the younger generation of neurosurgeons in the country whom I have seen at work have learned their surgery of the nervous system in this way. It has the double advantage of giving them a sufficient laboratory experience to enable them subsequently to pursue to the only place where they are likely to be solved some of the many problems which arise. It was fortunate in my own case and in that of my early co-workers in Baltimore that the Hunterian laboratory was available for this purpose.

But with all this concerning the technic of operation we must not forget that there is a technic of examinations, and that a thorough, orderly and well conducted neurologic study of a patient which demands the use of many instruments of precision necessary for tests of the special sense organs, is an art in itself. The examination of the eye is unquestionably the most important of all, and without the ophthalmoscope we would be quite helpless.

But in matters of localization the perimeter is of even greater value. In the series of papers (of which one or two are still to appear) on the "Fields of Vision in Cases of Brain Tumor," published successively with Drs. George J. Heuer, James Bordley, Jr., and Clifford B. Walker, we fell into an unquestioned error in relation to peripheral interlacing of the color fields. These observations were due to inexperience, and with the highly perfected methods developed by Dr. Walker corresponding field changes were no longer observed. They were due partly to an imperfect technic, but more to erroneous interpretations of the responses of inattentive and easily fatigued patients. Naturally, therefore, color interlacing was found to disappear after operation.

Our faulty observation need perhaps not be greatly lamented for it unquestionably served to reactivate interest in perimetry which before that time, in clinics other than ophthalmological ones, was rarely employed and then only for gross defects.

and less interest. It is possible in this way, doubtless, to make a greater number of these articles and hence, if they are to be sold, to insure a greater remuneration for someone, but it does not add to the happiness or satisfaction of the artisan. On the contrary, it deprives him of the supreme satisfaction of creating something from the bottom up in which, though an artisan, he comes to take artistic pride.

This is one of the underlying sources of the unrest and dissatisfaction of the laborer. There is something of this tendency in surgery today. Ten or more goiters or hernias treated, or appendixes removed, in a morning is not an uncommon record, but none of them is completed in a thorough-going sense by one person; the chief surgeon may spend but a few moments at the task—the more essential moments to be sure, and it is renumerative—but it puts him on the basis of that form of specialist whose sole task is to give the final adjustment to the carbureter as a succession of newly-assembled motor cars pass him. This, I do not believe, leads either to real satisfaction in one's professional work or to the greatest excellence in its quality.

It was said of Godfrey Kneller, who painted portraits innumerable, that he only did the faces—others in his studio particularly skilled at portraying hands and wigs and gowns filled in the rest of the picture. The quantity was great and the canvases show it.

THE BRAIN AND ITS ENVELOPES

Tumors.—I find that in the report of 1905 no mention is made of the number of patients who up to that time had been operated on. There had been very few—twenty-nine to be exact—with no instance of a really successful tumor extirpation. The principles of decompression were under discussion and at the time it was the common practice of physicians with a brain tumor suspect—and the diagnosis was not often ventured on—to prescribe a prolonged course of antisyphilitic treatment before considering an operation. It was not until two years later, be it remembered, that the Wassermann reaction was introduced, nor was it perfected by Noguchi until 1911; but even this did not entirely remove this source of delay, for as late as 1913 at the last International Medical Congress, Horsley felt impelled to protest urgently against this form of procrastination. There are other sources of delay which today must be combated, but fortunately this one has been eliminated.

In the early days, though palliative measures brought temporary relief to many, actual recoveries after tumor extirpation were exceedingly few. There were many discouragements even on the side of the temporary alleviation of symptoms, for many patients were in the terminal stages, a large proportion of them blind, or so nearly so that blindness ensued despite a thorough decompression. Then, too, the conditions found at necropsy in fatal cases were such as to make it seem that if cures were ever possible they would certainly be so few and far between as hardly to justify the expenditure of such labor.

Curiously enough, looking back on this early series, the first two patients who appear to have been actually cured, if one may use this term critically, both had cerebellar gliomas, and were operated on in 1908. One of them was a small boy of 13 years with a gliomatous cyst twice operated on, the last time three years later, with what appears to have been a successful destruction of the cyst wall. His functional recovery was perfect. He subsequently became captain of his school football team and now, nearly thirteen years later, is married, the father of a family, and regards himself as perfectly well. The other patient was a young man of 28, with a large glioma removed from the left hemisphere. Except for a slight impairment of vision in one eye on the recession of his choked disks, his recovery seems to have been complete, as may be appreciated from the contents of a recent letter sent me on the twelfth anniversary of his operation:

> . . . Being 39 years old, married and with two children beside other dependents, I did not feel as though I could be spared from home and so instead of enlisting in 1917 I went with the Bartlett Hayward Company who had contracts for making shrapnel shells. My particular work was making a part of the 155 mm. shell. I had to read micrometers to the one-thousandth of an inch, and if I had to depend on my left eye I could not have done it for it was already affected before the tumor was removed, as your old charts will show, but the other is normal and good enough for two. I was able to stand the hard work with the best of them though I had the "flu" and pneumonia as did lots of others. . . .

One would have thought that the initial successes would have been in extirpations of benign tumors like the endotheliomas or possibly acoustic neuromas. To be sure, the first reasonably successful operation for an acoustic neuroma—a case reported by Allan Starr—dates back nearly as far, namely, to 1909, and though the patient remains reasonably well the degree of functional recovery is by no means perfect, for though these recess tumors are benign they present exceedingly difficult surgical problems.

Doubtless in the earlier series many endotheliomas were overlooked, for, concealed as they are apt to be between the hemispheres with but little surface evidence of their presence, they are difficult to expose—at least I can only account for their present proportional prevalence by this assumption. It was not until 1910, shortly before the reading of my second paper, that I had my first really successful endothelioma extirpation with a perfect result, in a man who since that time has taken an active part in national affairs.

Though in 1910 there were only 180 tumor cases in the series, they nevertheless were beginning to appear in increasing numbers and have now become so many as to necessitate limiting the number under observation at a give time. All told, in the Baltimore series there were 330 cases up to the fall of 1912, and since then the series of of 735 cases observed at the Brigham Hospital to September 1 brings the number well above 1,000. One hundred examples of a given lesion each year provides as much material as can well be studied, and one must check the tendency to accept more than can be digested. It was far easier to comment ten years ago on 180 cases of presumed tumor than today on 1,000.

Brain tumors are common—far more so than I believed to be the case when this same statement was made fifteen and again ten years ago. The time has come when we can no longer afford to group them together as a class any more than we can group all abdominal tumors together and make a composite report on them which is at all worth while. We must take tumors in certain situations or, better still, tumors of certain kinds in certain situations, and give them special study. I have attempted to do this in a monogroph dealing with the acoustic neuromas, of which there have now been forty-five verified cases, and feel that such a group plan is the only way really to advance the subject. I shall hope ere long to follow it with a similar report on a series of some sixty verified endotheliomas which represent a particularly interesting type of intracranial tumor.

Tumor Classification.—It is an Irishism to say that other conditions sometimes resemble tumor more closely than tumor does itself. I mean by this that some of our most favorable examples of tumor are ones in which the classic symptomatic triad of headache, vomiting and choked disk is completely wanting, though all three would doubtless have appeared in time. On the other hand, many patients are admitted to the clinic with a presumptive diagnosis of tumor, of the presence of which, nevertheless, we are often extremely doubtful despite the full blown classic phenomena.

We have come, therefore, to classify the cases as (1) *tumors certified* (or verified) only when the diagnosis has been confirmed and the type of tumor identified by histologic examinations of tissue secured either at operation or necropsy;[9] (2) *tumors uncertified* when, though not histologically confirmed, the diagnosis is well nigh unquestionable; (3) *tumor suspects,* when it is a likely diagnosis. In this last group we keep a careful list also of a variety of conditions loosely designated as *pseudo-tumor* (though not in the meaning of Nonne), comprising states, whether so proved or not, other than tumor, but which have been sent to the clinic in view of a supposed tumor syndrome. The tentative diagnosis of some of these conditions, as other than tumor, may come to be verified subsequently at operation or necropsy, but mistakes in these borderline lesions are common and cases long classified as pseudo-tumor sometimes prove in the end actually to have been tumor cases, just as the case of an occasional patient with the diagnosis of "tumor uncertified" proves in the end to be other than a tumor case.[10]

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[9] We make one exception to this, namely, in the case of gliomatous cysts the character of whose fluid contents in about 99 per cent. of cases serves to verify the character of the lesion.

The following greatly compressed table will show something of the numbers and give a general idea of the situation of the tumors in my complete series up to the first of September.

TABLE 1.—AUTHOR'S BRAIN TUMOR CASES

Series	Forebrain			Pituitary Including Supracellar		Hindbrain			Totals
	Certified	Uncertified	Suspects	Certified	Uncertified	Certified	Uncertified	Suspects	
Baltimore.......	101	51	23	41	20	53	34	14	337
Boston..........	146	110	62	132	52	131	72	30	735
Totals........	247	161	85	173	72	184	106	44	1,072

It will be seen that of 1,072 cases classified in the tumor group, in 604, or about 60 per cent., the nature of the tumor has been histologically verified. It would lead us too far in a general address to attempt a classification of these verified lesions or to tabulate them according to their anatomic regions, though in view of their group predominance the tumors of hypophysial origin have been separately listed in Table 1. We must concern ourselves on this occasion more with the surgical than with the pathologic aspects of these conditions.

To give some general idea of the more strictly operative features of the work, the following figures of a consecutive twelve months of service ending Sept. 1, 1920, have been assembled for me. Of 182 patients admitted with a diagnosis of presumptive or possible tumor during this pereiod, the cross reference cards show that fifty-four were not operated on.[11]

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[10] This question of tumor classification I brought before the American College of Surgeons in an address last fall, and it is a matter which Dr. Percival Bailey has elaborated in some detail in connection with the series of cases observed during his sojourn as my assistant.

[11] These may be dismissed with the explanation (1) that three patients died shortly after entering the hospital, two of them with *tumor verified* at necropsy; (2) that twenty-three are recorded as *cerebral* or *cerebellar tumor*

The remaining 128 patients were operated on in 160 sessions with sixteen fatalities, giving an operative mortality of 10 per cent., a case mortality of 12.5 per cent. and a mortality of 15.5 per cent. for the seventy-seven patients for whom a tumor extirpation, partial or complete, was attempted. Considering the gravity of many of these procedures the percentage may be considered reasonably low, but it is capable, I am sure, of being reduced by one half with earlier and more exact diagnoses.

TABLE 2.—Table of Operations for Twelve Months Ending September, 1920

Character	Number	Fatalities
Forebrain:		
Subtemporal decompressions (palliative)	20	1
Subtemporal decompressions (disclosing tumor)	2	0
Osteoplastic exploration (cerebral)	5	0
Osteoplastic exploration (with decompression)	24	0
Osteoplastic exploration (with total or partial tumor removal)	24	5
Pituitary:		
Transphenoidal (partial removal of tumor)	20	1
Osteoplastic frontal exploration	8	0
Osteoplastic frontal exploration (total or partial tumor removal)	7	0
Osteoplastic temporal exploration	1	1
Cerebellar:		
Suboccipital exploration and decompression	25	2
Suboccipital exploration (with total or partial tumor removal)	24	6
Totals	160	16

The following additional notes of the fatalities should be given. One of them occurred in the series of twenty-two subtemporal decompressions for unlocalizable tumors. No examination was permitted and the lesion therefore remains permanently not certified.

Of the twenty-four attempts to remove cerebral tumors when exposed by an osteoplastic cranial resection there were five fatalities, four of them in the endothelioma group, as follows: of the gasserian ganglion, 1; of the falx, 1; of the mesial hemisphere, 2 (the death in one occurring several months after a total extirpation; in the other, an old man of 86 with epilepsy, it occurred from exhaustion due partly to a prostatic obstruction necessitating a suprapubic operation). The fifth fatality in this group followed the extirpation of a large glioma.

Of the thirty-six pituitary, or attempted pituitary, exposures operated on by various routes, two patients were lost, giving a mortality of 5.5 per cent. One of the twenty transphenoidal operations, an advanced case of acromegaly, died in forty-eight hours (necropsy refused). There were no fatalities in the fifteen transfrontal cases, but then, in a considerably smaller percentage in

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uncertified, one of these the third of the fatal unoperated cases; (3) the remaining twenty-eight cases are cross-indexed as *tumor suspects* and represent a great variety of conditions with such presumptive diagnoses as encephalitis, arteriosclerosis, aneurysm, cerebral syphilis, and so forth. In this group of twenty-eight there is only one case in which the lesion so far has been transferred from the tumor suspect to its proper group owing to verification of the lesion: a blind child with symptoms I had thought to be due to an infundibular tumor with hydrocephalus, but which proved to be due to a thrombosis of the superior longitudinal sinus.

this than in the transphenoidal group, the operation was successful in its purpose. The single attempt to deal with an interpeduncular tumor from the side was fatal, presumably from postoperative cerebral edema, a consequence of the dislocation necessary to secure a sufficient exposure.

Of the twenty-five suboccipital explorations for presumed cerebellar lesions, the two deaths occurred in cases of mistaken tumor localization, one that of a child with secondary hydrocephalus due to a bilateral papilloma of the choroid plexus, the other a patient with a large diffuse cerebral glioma.

Six of the twenty-four patients with cerebellar tumor succumbed after a more or less complete tumor extirpation; one of them three weeks after operation, following a fall causing contusion and hemorrhage; another a patient with an acoustic tumor who also had exophthalmic goiter and who died of weakness on her twenty-eighth day; another an acoustic tumor extirpation with death from inhalation pneumonia on the fifteenth day. The fourth patient died on the second day of respiratory paralysis after a partial removal of a glioma from over the fourth ventricle; the fifth died suddenly on the third day, after a thorough evacuation of multiple cysts due to a chronic arachnoiditis (pseudo-tumor, verified); the sixth patient died six weeks after an apparently successful extirpation, intact, of a tuberculoma.

The highest mortality, therefore, in this twelve months' series was in the suboccipital group of forty-nine operations with eight fatalities, giving a 15 per cent. mortality, or a 25 per cent. mortality for the cases in which tumor extirpation had been attempted. In this particular respect the record for the twelve months was a bad one, but the high percentage is due to the inclusion of the three patients who died some weeks after the operation from causes not directly attributable to it. Were one justified in eliminating these three cases the mortality would fall one half, which is about where it should be.

Needless to say, in this entire series there has not been a single case of wound infection. One of my assistants, who had served in the hospital for over two years, had seen a fungus cerebri only in pictures until he went to France with our hospital unit and met with this condition for the first time in the cases of cranio-cerebral injury to gunshot wounds.

Pituitary Disorders.—In my address of 1905 there is a footnote to the effect that conceivably a diseased pituitary body might some day be successfully attacked. How little was understood of pituitary disorders at the time can be appreciated from the fact that not until a year later (1906) was the first example of an infundibular tumor recorded in the Johns Hopkins series, and though a diagnosis of tumor with hydrocephalus was made, we had not the faintest idea of the location of the lesion. When this was disclosed long after at necropsy we had, even then, no inkling of the disturbances of metabolism the tumor had provoked.[12]

Today the diagnosis of so-called adiposogenital dystrophy, particularly when due to a supracellar tumor, would be made at sight by an undergraduate, for the clinical aspects of pituitary insufficiency when once pointed out prove to be as striking as those of cretinism or myxedema. Chagrined as we were by this experience, it nevertheless served a good purpose since it made possible an interpretation of the experiments undertaken the following year in the Hunterian Laboratory with S. J. Crowe and John Homans. For in the course of some experimental canine hypophysectomies, the counterpart of these clinical states, showing that they are due to a deprivation of pituitary secretion, was unexpectedly hit upon.[13]

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[12] This case, with one other, was reported under the title "Sexual Infantilism with Optic Atrophy in Cases of Tumor Affecting the Hypophysis Cerebri," J. Nerv. & Ment. Dis. **33**:704 (Nov.) 1906.

From this small beginning it has come about that hypophysial disorders are now recognized almost as often as those of the thyroid, and there is no reason to believe that one of these two glands is affected by disease any oftener than the other, though in view of the exposed seat of the thyroid the diagnosis may be read from afar.

In March, 1909, I first ventured to operate for a pituitary tumor in a case of acromegaly, employing a modified transsphenoidal approach to the sella, the result of abundant studies on the cadaver. The procedure was merely a further development of the operation by way of the nose, first used in its crudest form by Schloffer, the chief modifications making it possible to avoid nasal deformity and to lessen the likelihood of a postoperative infection. The case was a fortunate one[14] and led me to develop the operation by this route still further, though fully aware of its limitations, for it is only appropriate to those cases in which there is a wide dilatation of the sella.

At the time of my second address in 1910 there had been twenty cases under observation, and the following cautious expression of opinion was given:

> No one as yet would venture to assert that a tumor in the infundibular region can be totally removed, and inasmuch as pituitary headaches and pressure against the chiasm may be relieved by a local decompression or by the chance evacuation of a cyst, it would seem wise for the present at least, in view of the importance of the gland, to confine operative measures in most cases to the mere removal of the sellar base with incision of the glandular capsule—a local decompression.

A further statement expressing conservatism was made to the effect that "hypophysial tumors which have extended from the infundibular region into the cranial chamber proper must be treated by the same rules of decompression as would be applied to any equally inaccessible lesions elsewhere."

Time has shown that the former statement was unnecessarily cautious for in most transsphenoidal operations a considerable amount of tissue is removed. In the latter statement I was quite wrong, for a subtemporal decompression rarely benefits a patient suffering from pituitary headaches, for they are due to local rather than general pressure effects. Hence when an adenomatous gland has penetrated its dural envelope and invaded the cranial chamber, it must be surgically approached from above, if at all, by an osteoplastic exposure of the region.

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[13] Clinical Aspects of Hyperpituitarism, J. A. M. A. **53**:249 (July 24) 1909.

[14] Partial Hypophysectomy for Acromegaly, Ann. Surg. **1**:1002 (Dec.) 1909.

In a monograph on pituitary diseases, published in 1912, after a general discussion of the subject, my forty-three surgically treated cases were recorded. I had had but a single experience at the time with osteoplastic temporal procedures, such as Horsley had practiced, and none with the approach from in front by elevating the frontal lobes as originally proposed by Hartley and McArthur.

With our increasing familiarity with pituitary tumors, however, the cases with an indubitable infundibular lesion surmounting a more or less normal sella, which would preclude an operation from below, came to be so often recognized as to necessitate the employment of some osteoplastic measure, and the frontal procedure was given preference. Since my first case (Dec. 12, 1913), a considerable number of these operations, occasionally with a gratifying result, have been undertaken. Recently Heuer and Dandy in Baltimore and Adson in Rochester have warmly advocated a development of the lateral approach, first employed by Horsley. Unquestionably there is no one operation suitable for all cases, but I feel that the postoperative complications and high mortality, approximating 40 per cent., which so skilful an operator as Dr. Huer admits,[15] in view of the scant promise of permanent cure on recovery, will deter others from adopting this procedure.

In general terms, it is my present opinion that when there is a primary pituitary adenoma with enlargement of the sella and signs of implication of the chiasm, the operation of choice is a transsphenoidal one. Properly conducted, it is an operation of comparatively low mortality,[16] convalescence is a matter of a few days and the results are often brilliant, with restoration of vision—the main object of the operation—which may be astonishingly rapid. However, as Dr. Heuer points out, it is not an operation which is likely to be repeated with equal success (true enough of any operation) in case the adenoma continues to grow rapidly or has already broken down the dural barriers and invaded the cranial cavity. Under these circumstances, if an operation is to be done at all, it must be from above if there is to be any prospect of preserving or restoring vision, and this—an improvement in vision—not mere operative recovery, is our present criterion of a successful procedure.

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[15] Heuer, G. J.: Surgical Experiences with an Intracranial Approach to Chiasmal Lesions, Arch. Surg. **1**:368 (Sept.) 1920.

[16] At the time of writing my monograph on the "Pituitary Body" in 1912 there had been twenty-nine transsphenoidal operations with four deaths (13.7 per cent. mortality). By the time of the Weir Mitchell Lecture (J. A. M. A. **63**:1515 [Oct. 31] 1914) on "Surgical Experiences with Pituitary Disorders" there had been to May 12, 1914, seventy-four of these operations with seven fatalities (9.5 per cent. mortality). A review of the cases since then shows an additional eighty-three operations with eight fatalities (9.8 per cent.), making 157 transsphenoidal operations in all with a mortality of 12.1 per cent. for the entire series.

It is very doubtful, however, at an early stage of a primary intrasellar adenoma when the conditions are favorable for an operation through the nose, whether patients should be urged to, or would be willing to, submit to an operation from above, with its admitted high mortality and likelihood of complications. For it is accompanied by a wide exposure of the brain whose cortex may suffer injury as may indeed the chiasm itself, and the operation furthermore opens the dural barriers so that further enlargement of the growth finds ready access to the region we would wish to protect.

With primary infundibular tumors surmounting, as they often do, a small sella of normal proportions, the problem is entirely different, and I am led to favor, with some modifications, the unilateral osteoplastic frontal operation. In the thirty-four patients with operations of this type (forty-three in number) there have been only two fatalities, the second and third cases. However, in only seventeen of the thirty-four patients was the lesion disclosed, and it could not be satisfactorily dealt with in all of them. In 50 per cent. of the cases, therefore, the nature of the lesion remains uncertified.

In a few cases, nevertheless, the results have exceeded all expectations, and it has been possible to enucleate small solid tumors or to empty a cyst and subsequently to detach and draw out its collapsed sac. But I must confess that it is not an operation to be made light of, and I have occasionally added to the preexisting damage of the optic nerves. Indeed it has been necessary once or twice to divide the functionless one of the pair in order to dislodge the growth. In the case of the pharyngeal-duct cysts, a condition which would seem most favorable, the results have been disappointing, for the cysts promptly refill unless the wall is fully removed.

The fight for vision is the crux of these operations, and, after all, the question of proper methods we neurosurgeons must settle among ourselves, not by writing papers but by seeing one another's cases and attending one another's clinics. Meanwhile neurologists and physicians who have the decision to make may be comforted by the fact that the majority of these operations can be performed with a comparatively low mortality and a degree of symptomatic improvement which is constantly increasing.

Patients with disorders of the ductless glands, of the hypophysis in particular, show more or less characteristic mental attributes not necessarily deviating widely from the normal. I was inveigled once into writing an article on the subject, and whether from this or from some other beginning certain practitioners calling themselves endocrinologists have erected an extraordinary structure of symptomatic complexes based on meager evidence and a fervid imagination. This has reached its climax in a recent monograph by a psychiatrist, Laignel-Lavastine. There is no way apparently of checking these elaborations, which bear about as much relation to the functions of the ductless glands as did the phrenological imaginings of Gall and Spurzheim to cerebral localization. Epidemics of this sort from time to time hysterically sweep over Medicine, dying out in due course, the more quickly if unmolested.

Studies on the Cerebrospinal Fluid Circulation.—It is true of all intracranial operations that in the knowledge of ways and means of controlling tension lies the secret of surgical success. Intracranial over-tension is largely a matter of an increase in the fluid content of the chamber, whether it exists as free fluid in the ventricular and arachnoid spaces or as an edema, a state to which the nervous tissues are particularly prone. Though formerly the extensive withdrawal of fluid was looked on with apprehension, it has come to be an essential step in many cranio-cerebral operations, during the course of which the fluid, either from ventricles or arachnoid spaces, is not only thoroughly evacuated but may be permitted to drain away during the course of a long operation. There may be ways other than by external drainage of accomplishing the same thing, but to this we will return.

One of the problems which confronts the neurosurgeon is some method of dealing with the disturbances of cerebrospinal fluid outflow which lead to such malformations and disorders as spina bifida and "essential" hydrocephalus. In the latter condition, operations innumerable have been advocated since the earliest time, none of them proving to be based on correct principles if one may judge from their lack of success in practice. I have always cherished the view that most of these hydrocephalic conditions were produced by some failure of development of the points of escape for the fluid from the cranial chamber rather than, as was long supposed, from a fault or obstruction at the foramen of Magendie. The fact that in most cases the fluid could be withdrawn from the ventricles by a lumbar puncture, in other words, that it had access to the subarachnoid spaces, certainly seemed to favor this view.

Some early studies on hydrocephalus were carried out in the Hunterian Laboratory with Walter E. Dandy in 1910-1911. It was found possible not only to catheterize the aqueduct of Sylvius but to produce an experimental hydrocephalus by mechanically obstructing it. At the same time some early attempts were made to investigate the functional activity of the plexuses but we did not get very far with them.[17]

It became apparent that it would be necessary to carry our studies to the meninges if we were to find the most likely sources of obstruction. This work was undertaken by Lewis H. Weed in conjunction with Paul Wegeforth in the Surgical Laboratory during my first two years at Harvard. In the introductory notes to the series of reports on this work[18] a statement of the problems we had set ourselves to solve, so far as time would permit, is as follows:

> . . . Granting that the choroid plexuses are the chief source of the cerebrospinal fluid—and this has not been conclusively proved—is the process, as some believe, a transudation, or an actual secretion, or, as Mestrezat regards it, a mere dialyzation from the blood? What conditions activate and what conditions inhibit these choroidal glands? Have they an internal as well as an external secretion? To what primary diseases are they subject? How early in embryonal life do they secrete? Why does the fluid which they elaborate differ so greatly from that secreted by most other glands? Why are the cells so impermeable to the passage from the blood stream of drugs and of substances such as the bile pigments which, in conditions of jaundice, quickly stain all other body tissues and fluids?
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> Granting that the fluid thus secreted by the choroid plexuses leaves the ventricles and spreads over the brain and down the cord in the subarachnoid spaces, does it receive accessions from elsewhere, from the ependyma or from pituitary or pineal glands? Are there lymph channels in the brain, and if not, how does the central nervous system dispose of its products of tissue waste? If there are cerebral lymphatics do they discharge into the subarachnoid spaces and is the subarachnoid fluid therefore of the same character chemically, physically, and cytologically as the ventricular fluid? Why normally is the fluid practically limited to the subarachnoid spaces, and under what conditions does it become subdural?
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> Granting that fluid may escape by way of the Pacchionian granulations, is this the chief or only manner of escape? If an important avenue, why are these structures lacking in the lower animals and in the human infant? Are these granulations therefore pathologic processes, and if so what are their precursors? Are there other means of fluid absorption along the nerves by way of the lymphatics, and if so how important are they? How do the spaces

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[17] Dr. Dandy has recently reported the results of these and some more elaborate subsequent studies which he undertook independently. By an ingenious method he has been able to show quite conclusively that the fluid is elaborated by the plexus without the ependyma taking any part in it. Experimental Hydrocephalus, Trans. Am. S. A. **37**:397, 1919.

[18] Studies on the Cerebrospinal Fluid and Its Pathway, J. M. Res. **31**:1, 1914.

in the pia-arachnoid develop and do the choroidal glands mature and secrete before or after their formation? Are there faults of development at these meningeal outlets for fluid which can account for congenital cephalocele? Are there analogies in the fluid circulation of the eye to which we may attribute the disturbances of circulation of the intra-ocular fluids?

Weed, by adapting the principle of injection of nongranular fluids from which granules might subsequently be precipitated, showed conclusively that the arachnoid villi represent the points of escape for fluid which, by a process of seepage, enters directly into the pachymeningeal sinuses. It was, I believe, Wegeforth's proposal that in cases of hydrocephalus a series of direct punctures be made directly through the sinuses into the subarachnoid spaces, under the assumption that the puncture holes would become occluded by arachnoid, making thereby new and artificial villi through which fluid might escape.

Weed's continuation of these studies on his return to Baltimore and his demonstration of the manner of development of the fetal arachnoid spaces which I assume (though the point remains to be proved) to take place when the choroid plexuses first begin to secrete actively, certainly stands as the most important contribution to our knowledge of the meninges since Key and Retzius.[19]

If it is true that most cases of congenital hydrocephalus can be accounted for by a faulty development of the villi, the rational treatment is to reproduce in some way this channel of outflow either by direct drainage into one of the larger sinuses or by encouraging the formation of new villi in the manner Wegeforth suggested, rather than by an attempt to check the formation of fluid.[20] At least it is along these lines that our surgical efforts have tended of late years. Certainly no form of drainage into tissue spaces is effective, for by a curious property of the extracranial tissues when they are made edematous by cerebrospinal fluid leaking into them, a smooth endothelial-lined sac ultimately forms which is impervious to the further escape of fluid.

Such a procedure as Dandy has shown to be surgically possible, namely, the excision of the plexus from within the dilated ventricle, can hardly be expected to serve the desired purpose, for if there are any remaining fragments of plexus (and it is a complicated organ) the same degree of tension should recur so long as the ultimate outlets for the fluid are defective.[21] Possibly no more conclusive argument than Weed's more recent paper [22] could be given favorable to the view that imperfect formation or occlusion of the villi is one source, if not the most common one, of what has been called idiopathic (for lack of understanding of the process) hydrocephalus. By injecting a suspension of lamp-black into the cerebrospinal spaces of kittens, he was able to produce extreme degrees of internal hydrocephalus quite comparable to similar conditions seen in human infants.

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[19] The Development of the Cerebrospinal Spaces in Pig and Man, Contributions to Embryology No. 14, Carnegie Institution Publications No. 225, 1917, p. 116.

[20] In his more recent paper (The Diagnosis and Treatment of Hydrocephalus Resulting from Strictures of the Aqueduct of Sylvius, Surg., Gynec. & Obst. **31**:340, 1920), Dandy estimates that 66 per cent. of all cases of congenital hydrocephalus are due to Sylvian obstruction. If this is true the agency of obstruction to which we have devoted attention is less common than we had supposed.

At the present moment there are two or three new proposals before us which have a bearing on the cerebrospinal fluid and its spaces, and though admittedly still in an experimental stage, one or all of them may come in time to have considerable importance from a diagnostic as well as therapeutic standpoint.

In a recent paper, which has aroused great interest,[23] Dandy has put forth some very definite claims regarding the localizing value in cases of brain tumor, of what he calls *ventriculography*. This is, in other words, the taking of roentgen-ray plates of the cerebral ventricles after their fluid contents have been removed and replaced by air.

It is quite certain that in some rare conditions a more exact localizing diagnosis might be made in this way than in any other, and, in the case of tumors situated in silent areas above the tentorium which have led to dilatation as well as deformation of one of the ventricles, perhaps only in this way. That the procedure may be sufficiently developed and safeguarded so that it can be routinely utilized for this purpose is quite within the realms of possibility.

One particularly striking case has been included in Dandy's report, whereby an unsuspected tumor in the right occipital lobe was disclosed, though one must confess that it might have been capable of localization by earlier and more exact perimetry. However, as I have stated, the procedure is in an experimental stage and surgeons may safely leave it in Dr. Dandy's hands to work out more thoroughly its possibilities as well as its hazards, as he doubtless intends to do; and it would be highly desirable under these circumstances for every one who undertakes the procedure to notify its inventor of their experiences and particularly of the accidents. In this way only will he be able to establish the proposal on a safe basis acceptable to others, as all hope that he may be able to do. That there have already been a goodly number of fatalities, doubtless in the hands of people less expert than the author of the method, is well known. It will soon have a bad repute if so much is expected of it as is given in the author's conclusions, and if the surgeon is encouraged to believe that henceforth he will have less need of exercising his neurologic knowledge in localizing brain tumors.

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[21] Grave doubts have been expressed by workers in Carlson's laboratory as to the justifiability of the common assumption that the plexus is an active secretory organ and therefore that there is such a thing as a cerebrospinal fluid circulation (Becht, Frank C., and Matill, P. M.: Am. J. Physiol. **51**:1, 1920). If they are correct and we wrong, our entire theorem falls to the ground.

[22] The Experimental Production of an Internal Hydrocephalus, Contributions to Embryology No. 44, Publication No. 272, Carnegie Institution, 1919, p. 425.

[23] Dandy, W. E.: Localization or Elimination of Cerebral Tumors by Ventriculography, Surg., Gynec. & Obst. **30**:329, 1920.

Another procedure likewise in an experimental stage, though capable, it is hoped, of further development, is the diagnostic *puncture of the cisterna magna*, a procedure worked out in the Army Neurological Laboratory under Dr. Weed's direction during the war, and which has subsequently been warmly advocated by James B. Ayer.[24] When one realizes how loath the profession was to adopt Quincke's lumbar puncture as a more or less routine measure, one hesitates to say that a suboccipital puncture will not some day come to be as commonly employed. However, even a lumbar puncture is not without risk, as those are well aware who have seen patients with unrecognized cerebellar tumors die from respiratory paralysis soon after one has been made; and the risks of a puncture of the posterior cistern under similar circumstances would be infinitely greater. Hence this procedure, like the foregoing, had best be left in the hands of its sponsors until they can give us complete details not only of the method but of its diagnostic and therapeutic possibilities, and above all of its hazards.

But probably the most suggestive papers issued by this laboratory during its short life were those by Weed and McKibben [25] on the experimental *alteration of brain volume* following the intravenous injection of various substances in solution. They observed that, after the cortex was exposed by a trephine opening, the intravenous injection of a watery solution caused the brain to protrude through the

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24. Wegeforth, P.; Ayer, J. B., and Essick, C. R.: The Method of Obtaining Cerebrospinal Fluid by Puncture of the Cisterna Magna (Cistern Puncture), Am. J. Med. Sc. **157**:789, 1919.

25. Pressure Changes in the Cerebrospinal Fluid Following Intravenous Injection of Solutions of Various Concentrations, Am. J. Physiol. **48**:512, 1919; Experimental Alteration of Brain Bulk, ibid., p. 531.

opening, and contrariwise, that a hypertonic salt solution caused it to recede, sometimes to a very extraordinary degree.

That these observations had great possibilities of clinical application was immediately apparent to all, and Dr. Foley and others in my clinic have made it a matter of special study.[26] They found, in the first place, that it would answer almost as well to give sodium chlorid by mouth, and it is at times quite amazing to see what an immediate symptomatic effect, particularly when there is increased intracranial tension, this simple procedure may have.

That it is not entirely free from risk we have reason to know, but that it has great future possibilities of applicattion we nevertheless are encouraged to believe, though here again it is a matter of slow painstaking observation on the part of a few rather than indiscriminate observations on the part of many, which in time will establish its therapeutic and diagnostic possibilities and risks. As has been realized for a long time, the application of biophysical knowledge to the problems relating to the nervous system promises large returns, and Weed and McKibben's studies lie in this direction.

It would be premature to pass on the future rôle which any one of these three measures I have mentioned may come to play. Let us hope that they will not suffer a wave of wild popularity, to be cast off like von Bramann's callosal puncture, as good for nothing, simply because it could not do in others' hands everything and more than its author claimed for it.

THE SPINAL CORD

I have read over the general statement in my papers of ten and fifteen years ago regarding the surgery of the spinal cord, and though I might give many additional illustrations I do not know that there is very much to add to the general principles of these operations then described. There are more things, possibly, to retract than to add.

Infections.—In 1910 Flexner's serum for cerebrospinal fever had just been introduced and held the stage, but I fear that we are no further advanced in our treatment of other forms of meningeal infections than we were at that time, though proposals for irrigation and drainage of the meningeal spaces recur at more or less frequent intervals.

Great hopes were aroused, particularly from a prophylactic standpoint, by Crowe's discovery of the passage of hexamethylenamin through the choroid plexuses and its prompt appearance, after administration by mouth, in the cerebrospinal fluid. We did not know at the time that it appeared unchanged, and that only in an acid medium like the urine was it broken up with the liberation of formaldehyd. Crowe's observations, however, on the efficacy of the drug, particularly as a prophylactic, in experimental canine meningitis were nevertheless so convincing that we have continued with its use in certain conditions —in patients with basal fracture, before transsphenoidal pituitary operations, and so on. It certainly does no harm, though we may have been leaning on a broken reed.

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[26] Foley, F. E. B., and Putnam, T. J.: The Effect of Salt Ingestion on Cerebrospinal Fluid Pressure and Brain Volume, Am. J. Physiol. **53**:464, 1920.

In view of the fact that most of these pathogenic cocci are acid producers I have harbored the idea that in process of their multiplication enough acid may be given off by them to liberate a certain amount of formaldehyd in their immediate vicinity, without producing an appreciable change in the reaction of the fluid as a whole—in short, that in this way, even though the cerebrospinal fluid retains its faintly alkaline reaction, the growth of organisms may nevertheless be locally inhibited. We have attempted by physicochemical tests to demonstrate the truth or otherwise of this conjecture, without any really definite conclusions.

Tumors.—As stated before, there is no more satisfactory operation in surgery than the removal of an accurately localized endothelioma of the spinal meninges—no operation unless it be for some of the major trigeminal neuralgias, in which the transformation from a suffering and bed-fast invalid to a normal life is more like resurrection. One may imagine the elation which Horsley and Gowers must have felt in 1888 on the occasion of their epochal first case.

My experience with these enucleable tumors has not been great—only seven cases additional to the one mentioned in my 1905 address which had been reported elsewhere.[27] Of many things I have learned in fifteen years, one is to beware of reporting "cures." This man had a recurrence of his symptoms, and some years later, at a secondary operation, a growth was disclosed which to all appearances might have been the primary one. On this second occasion it was removed, together with the subjacent patch of meninges, and there has since been no recurrence. The lesson was thereby learned that it does not do merely to tilt these lesions out: their meningeal attachment from which they take origin must also be removed, and this is not altogether easy since the central point of attachment seems invariably to be at the point of emergence of a segmental nerve root.

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[27] Intradural Tumor of the Cervical Meninges with Early Restoration of Function in the Cord After Removal of the Tumor, Ann. Surg. **39**:935 (June) 1904.

It is astonishing how promptly it begins and how great a degree of functional recovery is possible in the flattened cords long subjected to pressure by such a tumor. What is more, the promptness with which this restoration sets in may be taken as an evidence of the delicacy with which the tumor enucleation has been conducted. The last of these patients in the series gave the history of having had a cancer of the thyroid removed in 1900 and in 1903 a cancer of the breast; consequently when her spinal symptoms began to appear a few years later they were naturally attributed to a metastasis. For this reason an exploratory operation was thought inadvisable, and she gradually became bedridden from paralysis. However, the results of a neurologic examination, coupled with the characteristic manner of onset of the symptoms, were so unmistakably in favor of a spinal endothelioma that an exploration was urged and the expected lesion found and removed. Voluntary movements in her previously paralyzed extremities were possible the afternoon of operation, and before her discharge she was walking with slight assistance and now, a few months later, goes about alone.

From these brilliant results, which are all too few, there is a long gradation of less and less favorable cases to the malignant growths at the other end of the scale. No sufferers so greatly tax one's sympathies as the victims of malignant spinal metastases, and in 1910 I find that I had even suggested "the deliberate transsection either of the entire cord or of the posterior columns alone, cephalad to the lesion." The alternative of pain or complete permanent paralysis is hard to face, for patient as well as surgeon, and it was one which I had shrunk from until a few years ago, though fully aware that the victims of a transsection the result of a crush usually lead an existence free from pain and surprisingly often take a curiously detached and philosophic view of their plight. This peculiar dispositional attitude of mind has seemed, in short, to be a mental attribute consequent on the abolition of all impulses from the lower body.

Much against my wishes, there was admitted to the wards in 1916 a woman suffering great pain from an obvious spinal metastasis, attributable to a breast amputation for carcinoma performed twenty-five years before. As her paralysis below the twelfth thoracic level was nearly complete, it did not seem possible that she could live long. For months her pain had been controlled by morphia, which seemed the only recourse. She, however, held out for months, courageous and cheerful though begging for some operative relief, until finally with many misgivings I divided the cord in the more accessible thoracic region well above her lesion. The results far exceeded both the patient's and my own expectations. She had no more pain whatsoever, narcotics were completely withdrawn, she gained in strength and color, acquired an automatic bladder control and lived in really great comfort, bodily and mental, despite what she knew to be in store for her. The end came six months later—the easiest and happiest end in a case of this kind of which I have cognizance.[28]

Akin to this, though less radical and advocated for less critical conditions, are other palliative measures which have been put in practice. Förster's posterior root division in cases of tabes, which is but a development of the old operation advocated for intractable neuralgias of amputation stumps, is one example: another is the procedure which Frasier and Spiller have described, of dividing the anterolateral columns alone, a procedure similar to that which I suggested in 1910, though based on more accurate localization of the pathway for pain.

Trauma.—What was said on this score from a surgical point of view in my first paper I do not think I can now improve on—the division into (1) the hematomyelias, (2) the total cross-lesions usually from fracture dislocation, and (3) the partial injuries, these being the only ones suitable for surgical intervention. The difficulty lies in distinguishing the three groups, and one must acknowledge that there is no sharp dividing line between them. In this respect, however, we will have learned much, chiefly through a study of the victims of spinal gunshot wounds in the late war, though it will probably be a long time before all the material is thoroughly studied. Placed as some physicians were, Colonels Sargent and Holmes, for example, at one of the important British bases, it was possible to secure specimens for later study of lesions of practically every segment of the cord in a series of cases which had been previously subjected to a thorough neurologic study. Madame Déjerine's opportunities with the spinal injuries occurring in the French army were almost as favorable, and she has already published some extraordinary observations concerning ossification of the muscles in areas below the segmental level of the lesion.

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28. Her death occurred while I was overseas, where the following letter was received from her husband. It may deserve quoting in view of the rarity of such procedure. ". . . After the operation she was brought home in May, and was enabled to enjoy the birds and flowers and go out in a wheel chair. She finally very slowly yielded to the disease, passing from us October 31. nearly a year from the time she entered the hospital. She was cheerful and patient during all her illness but beyond digestive troubles she had little pain and nothing acute. . . ."

Many remarkable and unknown facts were brought to light regarding the localization of function in the cord, but possibly the most immediately arresting were the studies of Henry Head and his co-workers.[29] Their observations have shown that, even when in its cross extent the lesion has been anatomically complete, the cord distal to the lesion is capable of resuming its reflex function to a degree hitherto unappreciated. This process, in the absence of infection, begins to set in after the third week, at which time the lower spinal reflexes begin to reappear. Hence the old conception of a permanent loss of deep reflexes with flaccid paralysis as an indication of a total transsection falls to the ground.

Though these observations are of great importance in advancing one's physiological knowledge, they do not necessarily affect in any apparent way our surgical procedures, except to influence surgeons to favor in every way possible the return of the so-called automatic bladder reflex which enables the unfortunate victims of these injuries to hold their urine for a few hours at a time.

This involves a problem relating to the care of these patients for which the war does not seem to have given a satisfactory answer, namely, the correct method of dealing with the bladder in the early stage of retention. The main desideratum, if one hopes to attain for his patient a subsequent automatic bladder control, is the avoidance of infection, and whether a constant drainage by catheter from the outset, repeated catheterization, suprapubic drainage, or the avoidance of any direct interference whatsoever and letting the bladder distend till it dribbles—which of these methods is best, no one so far as I am aware was ever able satisfactorily to determine, nor in this generation, will we again, let us hope, have so good an opportunity of finding out.

One thing the observations of Head have served to explain are the mystifying reports which have appeared in the literature from time to time regarding the restoration of function in a completely severed spinal cord after a laminectomy and suture. Unquestionably they were faulty observations to be explained by the reappearance of these lower spinal reflex movements which were erroneously interpreted as an evidence of transmission of impulses from above.

THE PERIPHERAL NERVES

The surgery of the peripheral nerves, other than cranial, belongs largely though not entirely in the domain of traumatic surgery, and as injuries of the larger nerves are often coupled with deformities and need what is called physiotherapy, together with some supporting apparatus in their after care, a bid for these lesions during the war was made by the orthopedists.

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[29] Fearnsides, Head and Riddock: Brain **40**: Pts. 2 and 3, 149, 1918.

It was calculated that fully 25 per cent. of all major injuries of the extremities were accompanied by a more or less serious involvement of important nerve trunks, and it makes little difference who cares for them—if an orthopedist, he must have a better neurologic training and operative technic than most possess; if a neurosurgeon, he must familiarize himself more than he is accustomed to with the mechanical correction of paralyses. The subject at all events received a great fillip during the war, and much has been learned and much unlearned regarding nerve sutures. Attention may be called to a few points which stand out clearly from the great number of published observations.

It has long been known that divided nerves, given half a chance, tend to reunite. Indeed, under circumstances when a purposeful section or avulsion of a nerve has been made it is well nigh impossible at times to prevent some degree of functional reunion. This the old-time peripheral operations for facial neuralgia made only too clear. What takes place has been more or less a subject of academic dispute between the supporters of the neuron doctrine and those of Bethe's school who believed in the possibility of a peripheral regeneration of axons. Though Bethe undoubtedly found axons in the peripheral segments, Langley's explanation of their presence was doubtless the correct one. In short, the proliferation of the cells of the neurilemma sheath is the only peripheral process and though this prepares the way for the axon, unquestionably the axis cylinder must grow down from the proximal segment.

From the great mass of material of the past few years the curious observation was soon made that certain nerves show a much greater tendency to perfect functional reunion than others. To take a single example, the prognosis after injury and suture of the musculospiral was far better than after a corresponding injury of the median. This appeared, moreover, to have no relation to the distance between the seat of the injury and the periphery, for a high radial suture was more favorable than a low median. The explanation for this which seems best to meet the facts is that the functional recoverability bears relation to the degree of purity, whether sensory or motor, of the nerve in question. Thus the musculospiral nerve has a great preponderance of motor fibers and consequently after suture there is less chance of motor axons finding their way down sensory pathways and the reverse than if the number of sensory and motor fibers were more nearly equal, as is true of the median.

This at least is the interpretation which appears best to fit the facts before us, though it is one which is difficult for me to believe as it seems a poor provision of Nature. Though we have little knowledge of what the nerve impulse really is, much less of the distinction between a motor and sensory impulse, there certainly must be some chemical difference between the two nerves which would lead us to assume a predilection of down-growing sensory axons for sensory paths and motor for motor.

Be this as it may, it is assumed that, when an imperfect functional result follows what seems to be a simple and perfect nerve-suture as so often happens after median nerve sutures, the majority of motor fibers have grown down into sensory sheaths to sensory end organs whose messages they are incapable of transmitting, and the majority of sensory fibers likewise to motor end organs.

This interpretation may be quite wrong, though as I say, it seems to fit the facts and, if correct, it would appear to favor such detailed operative procedures as Elsberg has described, based on Déjerine's studies of the cross sectional topography of bundles in the peripheral nerves. However, even if it is surgically possible to approximate a divided peripheral nerve, bundle for bundle, in the process of a nerve suture, which I doubt, there is something other than end-to-end proximity—and it is only a matter of millimeters—which attracts the wandering axon into some particular peripheral tube. Indeed one may see a perfect functional result when an actual (though small) gap has been left, or contrariwise, an imperfect result with the most painstaking approximation.

One thing has been made clear beyond question, and that is the supreme desirability of bringing the two ends of the severed nerve together without the interposition of a bridge, and ingenious ways and means of accomplishing that have been devised, not only by stretching and by mobilization of the nerves, but by shortening the gap in case of great loss of substance by retaining the limb in a position of acute flexion until union has taken place.

Losses of substance of several inches can be overcome in this way, and it has meant the abandonment of the pre-war ideas regarding tubularization and implantations as a means of overcoming gaps—procedures which by increase in scar formation doubtless served to defeat the very object they were intended to serve. To be sure, there have been some remarkable recoveries, particularly in musculospiral nerves after the implantation of nerve autografts,[30] and Nogeotte, whose experimental studies have been extensive, favors the implantation, if one must be made at all, of a section of nerve from another source (homograft), which has been fixed in alcohol, but of this I have no experience.[31]

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[30] Madame Déjerine informed me of a perfect result in a French officer in whom a gap of 7 cm. was thus bridged.

Unquestionably, interest in all these matters aroused by the war has turned the attention of many of our younger surgeons with renewed interest to some of the neurologic problems of surgery. Brief courses were given in New York, Philadelphia and St. Louis, largely under the direction of Drs. Elsberg, Frazier and Sachs—which served further to concentrate attention on the subject and to give certain medical officers, whose inclinations lay in this direction, some general ideas of traumatic neurosurgery.

Excellent as all this was, it is a far cry from traumatic to pathologic surgery, from doing a peripheral nerve suture to a trigeminal neurectomy, from the repair of a cranio-cerebral injury to the removal of a brain tumor. Then too, the lack of personal responsibility about one's army patients— a responsibility entirely assumed by the Government—coupled with the difficulty or impossibility of learning of one's end results, did not inculcate in many medical officers the fastidiousness of clinical study or operative technic which is a *sine qua non* of success in civil surgery and above all of civil neurosurgery.[32]

A National Institute of Neurology.—Though as a matter fact it was the first time that such a special line of work as is represented by neurosurgery was recognized by an Army Medical Corps, the department was not utilized to its full capacity for many reasons, not the least of which perhaps was the old chauvanistic idea that he who is called a general surgeon must do equally well all things surgical. Nevertheless, the desirability and need of specialization in military medicine more than in any previous war came to be accepted as a principle, and in some divisions of the service where it obviously could prevent wastage of man power, it played an important rôle.

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[31] The conclusions of G. Carl Huber, Dean Lewis and others (Repair of Peripheral Nerve Injuries, Surg., Gynec. & Obst. **30**:464, 1920) in relation to the bridging of gaps seem to be very much in accord with those of Nogeotte.

[32] I have not touched on the *surgery of the cranial nerves,* as this address is already too long. There are many interesting and important aspects of this topic, though possibly the surgery of the *trigeminus,* and of trigeminal neuralgias in particular, overshadows all else in this particular domain.

I have made these major neuralgias the subject of a recent study, and the results, given a year ago in the Hatfield Lecture before the College of Physicians of Philadelphia, are in process of publication. It may suffice to say that the modern operation of sensory root avulsion, whoever may deserve the main credit for evolving the procedure, is an operation practically free from risks and with a mortality lower than that of almost any other so-called ("major") operation in surgery.

In my own series, now comprising 348 cases, there have been only two fatalities, the ninth and thirtieth cases, both of them dating back to the days when efforts were made to remove the ganglion together with the root in their totality. There have been 318 consecutive cases without a fatality. There is no other operation in surgery which can so completely relieve such an incapacitating malady and with such slight risk. There are possibly 5 per cent. of the cases which are not cured, cases in which the diagnosis was incorrect and in which the facial pain, supposedly trigeminal, is really of some other origin.

Those of us, however, who had the overseas responsibility for the care of such injuries of the nervous system as would incapacitate soldiers for further service, aware of the fact that these men would be the last of all our wounded to recover, and even then very imperfectly, were disturbed by the lack of coordination of neurologic interests which possibly the scramble during our short period in the conflict made inevitable. Since most of the surgeons delegated to the neurosurgical subdivision had only a meager neurologic training and the neurologists were all occupied in the important field of psychiatry, none of us got as far with our neurologic problems as if we had been organized together.

Cognizant of all this, Drs. Salmon, Schwab, McCarthy and myself, after many conferences, were unanimous in the feeling that some more effective organization should be brought about and that centers should be established where psychiatrist, neurologist, neurosurgeon and neuropathologist could be brought together, and where functional as well as organic cases could be seen and studied and treated conjointly for the benefit not only of the soldier but of neurology itself.

This is all past history which I have gone into more fully on another occasion,[33] but I touch on it again for the reason that out of these conferences there developed the idea of a National Institute of Neurology. The four men whose names I have mentioned agreed to withdraw from the pre-war engagements to take full-time positions in such an institute, provided the necessary funds could be secured for its establishment on a generous national scale. No one could possibly have been better fitted to launch and direct such an institute than Colonel Salmon, whose interests largely lie in the sociological aspects of neurology. It was hoped that under him, as subordinates, a neurologist, a psychiatrist and a neurosurgeon might each have a hospital ward for their special cases, and that the neurological laboratory, which under the direction of Major Weed in Baltimore had done such admirable work during the war, might be taken over intact. It was assumed that during its first few years this institute would be entirely

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33. Concerning the Establishment of a National Institute of Neurology, Am. J. Insanity **76**:113 (Oct.) 1919.

at the disposition of the Government and the Army for the care of the neurologic cases which, as I have said, remain the last precipitate of the war injuries—cases which now, alas, are widescattered.

The venture, proving a success as it almost certainly would have done, it was anticipated that with the war work finished it would become a national center, an institute and hospital, for the study of disorders of the nervous system—a central rallying place where psychologist, psychiatrist, neurologist, neurosurgeon and experimental pathologist could work in cooperation—a training ground as well for their successors who for the first time in any country would be given an opportunity, after graduation, for a broad general neurologic training.

This, gentlemen, is by no means an idle dream. The situation two years ago was most favorable for the inauguration of such a movement, a movement whose ultimate objects were to bring intimately together all of those who from one aspect or another are attacking problems relating to the diseases of the mind and the nervous system. There is nothing which is possibly of more vital concern to the nation than a knowledge of the mental disorders to which its citizens are subject. There are few things relating to the health and well-being of the community more neglected. For a single imperfectly endowed clinic for the study of mental disorders probably twenty institutes for the study of cancer, of infectious diseases and so on have been established, whereas the insane forgotten in our asylums and those with nervous and mental disorders who helplessly frequent our clinics and outpatient departments out-number all other patients by far.

This project may seem far removed from neurologic surgery, but I have an instinctive feeling that some day such an institute on a scale as large as the recently founded Institute of Hygiene will come to be established. When that day arrives, the neurosurgeon will of necessity be a participant, and from the contacts therein possible will come to have the proper neurologic training essential to the success of his particular field. He will need a preliminary general surgical training, but he will nevertheless have to be a product of a school of neurology in a broad sense, such as we do not now possess, nor any other country.

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In the preamble to this address regarding specialization in general, my hearers may have been aware, between the lines, that I was reading my own confession into what was written. It is true, for despite a reasonably broad surgical preparation, I confess to have climbed into neurology by the outlying route described. It is for this reason that I thoroughly understand how essential it is for the neurosurgeon of the future, if he is really to do justice to this new specialty, to have a far better grounding in clinical neurology and psychiatry than I have had, for he must make his own diagnoses and must have an intimate knowledge of neuropathology if he is to know what conditions are amenable to surgical therapeutics.

Many people are eager to enter this field of work. Hardly a month passes without some well established surgeon writing to ask whether his assistant may be given some instruction in "brain surgery" and can we take him. We are glad to do all that is possible. "Yes, he attended lectures in neurology as an undergraduate, and then there are colleagues who will make his neurologic studies and use the ophthalmoscope for him." He comes and spends a short time as an onlooker and returns to his clinic a neurosurgeon.

It will be a bad thing for neurologic surgery when it becomes fashionable. Gynecology has already suffered from this, and orthopedics is in the way to follow. Tendencies in this direction, to my despair, are apparent. Glad as I am to feel that the importance of the subject as a special line of work is becoming recognized, and confident as I feel that the day will come when professors of neurology in our schools will have had a surgical training just as the present-day gynecologist and orthopedist must have, nevertheless the way to bring about this desired end is not through the surgical operating room alone, but by the slow process of the neurologic clinic and the laboratory. Let us hope that some day in a national institute, such as I have described, the all-round training which is essential may be properly acquired by medical graduates desiring to enter this field.

But for its own good, I pray that neurologic surgery may never get so far from the home of general Medicine and its immediate parent, surgery, that there will be any estrangement or any possibility of its being shut out of doors when the time comes for its return.

ABDOMINAL NEUROSTHENIA *

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Among the large number of so-called neurasthenics, there is a group in whom symptoms relating to the digestive apparatus largely dominate the clinical picture. This group has been classified by a number of writers as "abdominal neurasthenics" and the digestive troubles have been attributed, very properly in most cases, to the loss of tone in the muscular and chemical activities of the digestive apparatus secondary to the neurasthenia. As a result, treatment of the abdominal disturbances has been largely symptomatic, and investigation has usually stopped short after the examination of one or two test meals and stools have revealed nothing of marked significance.

With the aid of stomachics, sedatives, cathartics, irrigations, and other procedures, the digestive tube has been used merely as a machine for forced feeding in the attempt to regain the general strength and, in conjunction with the various other therapeutic measures, to restore the nervous balance.

A small series of cases from this group has been studied with great interest during the last six years and the findings both from investigation and from the results of therapeutic effort indicate a new line of attack which is successful in selected cases.

For brevity's sake a few characteristic case histories will be summarized and permissible deductions drawn from them.

REPORT OF CASES

CASE 1.—*History.*—Miss Bessie C., 33 years old, was well until 12 years of age. Since that time she had never been robust. She had had various children's diseases. For years she suffered from marked constipation, alternating with cramps and diarrhea. In 1910 she was operated on for "chronic appendicitis." The appendix was not found, but a large lymph gland was removed from the cecal region and was reported to be tuberculous. Since 1913 onward she had had irregular attacks characterized by loss of consciousness, followed by abdominal pain, nausea, vomiting and marked prostration. She became a chronic invalid, spending much of her time in bed. She could assume no regular duties in life, because of headaches, lack of stamina and abdominal disabilities. She was seen by many consultants who labeled her "neurasthenic and hypochondriac." She became progressively worse.

Physical Examination.—In the fall of 1916 she was placed in the care of Dr. Malcolm Goodridge. He found that she was of small size, slender, fairly well nourished and that she had good color. (She had just finished a three months' stay at a sanatorium.) Other findings were hemoglobin, 100 per cent.; red cells, 6,187,000; white cells, 10,000; polymorphonuclears, 70 per cent. Head, mouth, lungs, heart and urine were negative. Stomach, 100 c.c. retention 3 hours after 200 c.c. meal; achlorhydria. Stools were negative. The abdomen was somewhat distended with gas, and showed the scar of the appendix operation. There was a fairly marked tenderness over the right iliac region with gurgling of gas on pressure. There was also tenderness in the right hypochondrium near the ninth cartilage and just to the right of the median line in the epigastrium. The stomach was considerably enlarged.

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* Read at the Forty-Sixth Annual Meeting of the American Neurological Association, New York, June, 1920.

Roentgenograms revealed a large "fish-hook" stomach with the greater curvature below the brim of the true pelvis; the duodenum fixed high up under the liver with sharp angulation between the first and second portions and with distortion of the cap. The cecum was dilated and descended into the true pelvis. The hepatic flexure was fixed high and angulated near the duodenum.

Consideration of the clinical history and the objective findings, especially the enlarged stomach with retention, the high fixation and distortion of the duodenum, the high fixation and kinking of the hepatic flexure with marked dilation of the cecum and stasis in the ileum, led both Dr. Goodridge and myself to feel that the anatomic distortions caused the abdominal symptoms and that the neurasthenia had resulted from the oft repeated pains and continued toxic absorption.

Treatment and Course.—She was operated on Oct. 20, 1916, under ether anesthesia. A transverse right rectus incision was made 4 cm. above the umbilicus. The findings were enlarged stomach, freely movable, reaching well over into the right flank, then turning backward, upward and inward to the place where the summit of the duodenum was held fast to the proximal third of the gallbladder by a double fold of peritoneum which kinked and compressed it. This double fold passed over the duodenum down to the hepatic flexure, which it also held high and kinked. Neither duodenum nor flexure could be displaced downward. This double fold was divided transversely and the duodenum and hepatic flexure mobilized downward so that kinks and compression were relieved. The gallbladder was normal except for attachment of the double fold. Adhesions between the omentum and the scar of the previous operation were separated. A small retrocecal appendix was found and removed. There were no signs of tuberculous infection in the ileocecal region. A Jackson's membrane of moderate size which kinked and compressed the ascending colon was divided and the colon mobilized. The wound was closed and she was returned to bed in good condition.

Recovery was uneventful and comfortable. She remarked that a "dragging sensation" always present in the epigastrium had disappeared. Healing was by primary union and skin sutures were taken out on the sixth day. She took food freely and with relish after the first few days and without her customary pains and flatulence.

Two months after the operation she reported she had had no abdominal disturbance and had gained much in weight and color.

Sixteen months after operation she reported that she never had any discomfort after food unless she was extremely tired; she was troubled with gas only at rare intervals; she could eat all kinds of food and was moderately constipated. During this interval, for several months continuously, when her father and mother were both ill, she managed the household and helped with the nursing, doing far more in the way of continuous physical, mental and emotional effort than she had ever been able to do in the preceding six years.

At the end of two and three-quarters years she reported that her digestion was absolutely normal and her strength and capacity for steady work "decidedly" increased. She had some headaches and occasional constipation. During the last six months of this period she had felt better than at any time in years.

Case 2.—*History.*—Mr. W. T. J., Jr., 27 years old, was well until he was 26 years old, when he developed a marked nervous breakdown in the fall of 1916. In January, 1917, he had influenza twice, followed by profound neurasthenia. After two months of quiet outdoor life in the South he improved considerably. He then entered the Plattsburg training camp, but after a month was discharged because of "recurrent neurasthenia." He was then carefully examined by Dr. Walter Niles.

For over a year he had been much troubled with belching and flatulence, which was not accompanied by pain. His appetite was good and his bowels moved daily. Often he had a sensation of weight after eating. His chief complaint was dull headache, loss of memory and initiative, profound lassitude, mental depression and attacks of hysterical weeping. He was incapable of carrying on his work as a lawyer.

Physical Examination.—He was a tall, slender, dark skinned, sallow man. Examination of the head, throat and chest was negative. The blood pressure was 144 mm. The abdomen was normal in contour and mobility and was moderately tympanitic everywhere. The sigmoid was slightly tender. Slightly to the inner side of the right anterior superior spine and about 4 cm. below its level was a localized spot of extreme tenderness, associated with muscular spasm. To the right of the median line in the epigastrium was an area of definite but moderate tenderness.

Roentgenograms demonstrated a fish-hook stomach with the antrum well over to the right of the spine. It showed no deformity in outline and peristalsis was vigorous. The stomach was empty in three hours. The first portion of the duodenum was of average size, with smooth edges and no sign of ulcer. It was fixed high. Eight hours after eating there was slight retention in the ileum. Thirty-six hours after eating 90 per cent. of the meal had been discharged. The cecum was deep in the pelvis, from which it could not be dislodged, and it was tender. The urine was clear, acid, specific gravity 1.029, with a faint trace of albumin and no sugar. Microscopic examination was negative. Findings of the stool were negative; gram-positive bacteria, 25 per cent.; gram-negative, 75 per cent.

Treatment and Course.—He was operated on Nov. 27, 1917, under ether anesthesia. A transverse right rectus incision was made about 5 cm. above the umbilicus. The findings were: Stomach and pylorus free from ulcer or inflammation, duodenum fixed high to the proximal 4 cm. of the gallbladder by a double fold of peritoneum which caused definite kinking of the duodenum at the junction of the first and second portions. The gallbladder was normal except for the fold running to the duodenum. The liver was normal. The ascending colon was compressed by a Jackson's membrane. The cecum was nonadherent, dilated and had attached to it an appendix about 8 cm. long, irregular in caliber and with one considerable enlargement (fecal concretion) at about its middle. The remainder of the abdominal contents were normal.

The procedure was: Division of the duodenal membrane, mobilization of the duodenum downward (5 cm.) with whipstitching of the raw edges; division of Jackson's membrane with release of the ascending colon; appendectomy, with inversion of stump; closure of wound by layer sutures.

Recovery was uneventful, and he rapidly regained his health. He joined a naval cadet camp and went through the hard training without the least troube. His various neurasthenic and hysterical symptoms disappeared within a month of his operation and have never recurred.

On Jan. 24, 1920, he reported that he had been back at business since his discharge from the navy, that he was full of energy and initiative all the time, was never tired and felt better than ever before and had gained 30 pounds.

Case 3.—*History.*—Mrs. T. N., 39 years old, as a child had children's diseases. She had also been subject to attacks of abdominal pain at irregular intervals since childhood, chiefly in the right lower quadrant. These pains were of short duration, but were prolonged by walking. Five years ago these attacks became more severe. The last one occurred in June, 1917. Her periods had always been normal. She had two children, 10 and 8½ years old. Her appetite was fair, food caused no pain, but she had much flatulence, and was only occasionally constipated. She suffered frequently from severe headaches; tired out quickly and completely and was unstable in her nervous system and self control. She had become progressively worse during the last two years.

Physical Examination.—She was a tall, thin, sallow, irritable woman, weighing 110 pounds. Examination of the heart, lungs and urine were negative, as also was bimanual pelvic examination. The abdominal wall was flat, minus subcutaneous fat, and moved normally in respiration. The sigmoid was spastic and moderately tender to pressure. Pressure along the colon upward and to the right caused much gurgling of gas, but no pain until the right ninth costal cartilage was reached, where there was localized tenderness. The cecum was much distended. There was extreme tenderness over an area 2 by 1 cm. about half way from the right anterior superior spine to the umbilicus. As her appendix had to be removed, it seemed unnecessary to subject her to the trouble and expense of a barium series of the gastro-intestinal tract, as complete exploration could be done when the appendix was removed.

The diagnosis was chronic appendicitis; probable Jackson's membrane.

Treatment and Course.—She was operated on Dec. 4, 1917, under ether anesthesia. A transverse incision was made of the right rectus, 2 cm. above the umbilicus. The findings were: The stomach was greatly distended and crowding over into the right flank; there was no ulcer. The duodenum was kinked and held high by a short double fold of peritoneum running from it to the proximal end of the gallbladder and also by firm, thick adhesions running to the undersurface of the right lobe of the liver, 2 cm. to the right of the gallbladder. The liver and gallbladder otherwise were normal. The right kidney was freely movable. The appendix was pathologic, running upward and inward behind the cecum. Jackson's membrane extended from just above the cecum to the hepatic flexure, where there was a thick, short band, kinking and compressing and holding the flexure high. The uterus was normal. The right ovary was quite fibrous and had a firm nodular growth at its hilus. The left ovary was one cyst.

The following procedure was used: Division of membrane and adhesions binding duodenum to gallbladder and liver; mobilization of the duodenum downward 4 cm.; raw edges whipstitched; appendectomy, with inversion of stump; division of Jackson's membrane with mobilization of the colon; double oophorectomy, one ovary being a cyst and the other showing a probable beginning malignancy. The wounds were closed by layer sutures.

Convalescence was uneventful; the wounds healed by primary union and the patient returned home on the nineteenth day. She was given endocrine therapy from the fourth day after operation.

After five months she reported that she was feeling well and was slowly gaining weight. After eighteen months she was maintaining her improvement in color, weight, digestion, strength and capacity for sustained effort. She has recovered her nervous balance.

CASE 4.—*History.*—Miss Jean W., 17 years old, was said to have had typhoid when 3 years old and since that time had been subject to bronchitis. In 1910 she was operated on for hernia and had not been well since, suffering from severe headaches, anorexia, constipation, nervous depression, backaches, cold, clammy hands and feet, insomnia and entire lack of stamina. She was troubled with constant flatulence, by a sense of fulness after eating small amounts of food and by pain two hours after eating. Her periods were regular, profuse and painful, necessitating rest in bed.

She was studied by Dr. F. S. Meara, who reported the following results: Blood: hemoglobin, 81 per cent.; color index, 0.8 per cent.; red blood cells, 4,740,000; white blood cells, 6,900; polymorphonuclears, 45 per cent.; lymphocytes, 46 per cent. Urinalysis: The urine was acid; the specific gravity was 1.018; there was a faint trace of albumin and no casts. The stools were negative; gram-positive, 15 per cent.; gram-negative, 85 per cent. Lungs and heart were negative.

She was tall for her age, thin, and fairly nourished. The abdominal wall was thin and retracted, but moved normally with respiration. The stomach was enlarged down to the umbilicus. The sigmoid was spastic and moderately tender. There was distinct tenderness and resistance just to the left of the median line about 3 cm. above the navel; also at a point 6 cm. within and 2 cm. above the level of the right anterior superior spine. Otherwise the findings of the abdominal examination were negative. Her hands and feet were cold.

Roentgenograms showed: Marked gastroptosis, the greater curvature being below the sacral promontory; six hours' retention of barium; no sign of ulcer; retention in the duodenum with signs of obstruction at the duodenojejunal angle. The appendix visualized early, segmented and retained the barium throughout the period of investigation (forty-eight hours). The colon was slow in emptying, the cecum was club-shaped and showed retention. The ileocolic valve was incompetent.

Treatment and Course.—Operation was advised and accepted on the basis of the obvious disturbance of the appendix and duodenojejunal angle. The operation was performed May 30, 1918, under ether anesthesia. A transverse incision of the right rectus was made just above the umbilicus.

The findings were: The stomach was normal except for enlargement and adhesion between the pyloric antrum and the transverse colon. Pylorus and beginning duodenum were normal, except that they were dilated. The duodenojejunal angle was pulled upward and to the left by a strong peritoneal fold which caused marked angulation and obstruction. There were several enlarged lymph glands among the superior mesenteric vessels near the angle. A broad thick Jackson's membrane enveloped the cecum, ascending and beginning transverse colon, causing compression and kinking. The appendix was 10 cm. long, segmented and nonadherent. There was a calcareous gland in the ileocecal angle. The pelvic organs and kidneys were normal.

The following procedure was used: Plastic release of the duodenojejunal angle which came down 5 cm.; removal of the lymph gland for pathology; release of the colon from Jackson's membrane; appendectomy, with inversion of the stump; removal of the calcareous lymph node. The wound was closed by layer sutures.

Recovery was uneventful except for a stitch abscess in the skin. On the twenty-third day she left the hospital feeling better than for months. The lymph nodes were reported as showing simple chronic inflammation. Five months later her physician reported great improvement in every way. Twenty-four months later she reported marked improvement in appetite and capacity to take general diet. She was occasionally troubled with gas and constipation. Her capacity to do things was greatly increased.

SUMMARY AND CONCLUSION

These four cases, briefly summarized, were selected from a larger series to illustrate varying phases of the problem.

The patient in Case 1 had had a prolonged history of abdominal pain and digestive disturbance, on which the neurasthenic condition had been engrafted and finally became the predominating feature in the minds of many of the consultants. The patient in Case 2, who had had little digestive disturbance beyond flatulence and that without constipation, suddenly developed a neurasthenia, apparently idiopathic, which was greatly increased by influenza. For a year he had had various treatments without result. The patient in Case 3 also had few symptoms relating to the digestive tract, except flatulence and occasional constipation. Her neurasthenia came on gradually during several years, was associated with lack of mental balance and probably with disturbance of the endocrine system. The patient in Case 4 had never been robust and developed a profound neurasthenic condition following a simple herniotomy. In her case the neurasthenic and abdominal symptoms developed simultaneously and were about equally prominent over a period of eight years.

All four of these patients had been under the care of competent internists for from one (Case 2) to eight years, and were given repeated rest cures and other treatment, under which they improved only to slide back on attempting to take up their duties in life. Finally a painstaking examination of the digestive apparatus indicated what proved to be, in these cases, the chief underlying cause of the illness. The nerve exhaustion was apparently secondary to the absorption of toxins from the digestive canal, and it is my belief that the formation of these toxins largely resulted from mechanical interference with the normal motility of the digestive canal.

It is worthy of notice that in each case there was a high obstruction and a low obstruction. The high obstruction resulted from kinking and compression of the duodenum by a Harris band from gallbladder to duodenum in Cases 1, 2 and 3, and from kinking of the duodenojejunal angle in Case 4. The low obstruction in each case resulted from constriction and angulation of the ascending colon and hepatic flexure by a Jackson's membrane. In each case the appendix was definitely pathologic.

In this type of case, examination of the gastric contents seldom gives any information of value except as to the presence or absence of retention. Likewise, examination of the stools seldom gives information of value.

The diagnosis depends on the manual examination of the abdomen, showing tenderness over the appendix, ascending colon, hepatic flexure, gallbladder and pylorus or duodenojejunal angle, or showing distention of the cecum, or distention and ptosis of the stomach or dilatation of the duodenum. These may occur in varying combinations.

These findings are verified, amplified or modified by the roentgen series. A series to be of any value must be made by a man skilled in determining the times, attitudes and numbers of plates to be taken, and also in interpreting the plates and the results of fluoroscopy, which is one of the essential elements of a proper roentgen examination.

When an accurate diagnosis is made, a properly planned surgical procedure usually gives a prompt and permanent relief to the digestive disturbances and rapid recovery from the associated neurasthenia.

The obvious deduction from the facts presented is that a complete examination should be made of the digestive tract of every neurasthenic. In a certain proportion of cases surgery would offer a short cut to recovery.

A CASE OF CEREBRAL POROSIS WITH A HISTOPATHOLOGIC REPORT *

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In cerebral porosis the brain tissues contain a large number of cavities or cysts combined with minute holes generally described as "état criblé." Unlike other types of cavities, such as porencephaly, Marie's [1] lacunae, or Grasset's [2] progressive lacunar cerebrosclerosis, both cerebral porosis and "état criblé" are so far devoid not only of clinical but also of pathologic significance, being generally considered as artefacts, as accidental postmortem findings, in short, as cadaveric changes. One of these conditions, the "état criblé," has been discussed by me [3] at some length elsewhere, while the histologic study of the present case shows that cerebral porosis also possesses certain essential pathologic features which are due to intravitam changes and cannot be looked on as mere artefacts.

REPORT OF A CASE

A woman, 24 years old, born in Finland, entered Dr. Arthur Byfield's medical service of Cook County Hospital, March 26, 1920, in a semistuporous condition. Thirteen days previously she had been seized suddenly with severe abdominal pain accompanied by nausea and vomiting. This condition persisted for twelve days and greatly weakened the patient. She was able to be up and around, but was too weak to do her housework. On March 25, that is, on the twelfth day of her sickness, while taking a Turkish bath, she became "feverish" and so ill that she had to be removed to the Cook County Hospital. According to her family physician, she had not menstruated for six weeks.

Examination.—The patient was apathetic, but could be easily aroused and answered questions intelligently. The skin was dark bronze, the conjunctivae and sclera were yellowish brown, the nares were covered with dried blood, the neck was slightly rigid and the abdomen was tender throughout and rigid.

The tongue was heavily coated; marked pyorrhea was present. The respiration was labored, but there were no findings in the lungs or heart.

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* From the pathology laboratories of Cook County and Psychopathic Hospitals, Chicago.

[1] Marie, Pierre: Des foyers lacunaires de désintégration et de differents autres états cavitaires du cerveau, Rev. de méd. **21**:281, 1901.

[2] Grasset, I.: La cérébro-sclérose lacunaire progressive d'origine arterielle, Semaine méd. **24**:329, 1904.

[3] Hassin, G. B., and Bloch, Leon: Trichinosis Complicated by Encephalitis, Med. Record **91**:537 (March 31) 1917.

The reflexes (abdominal and knee) were absent; the Babinski and Gordon reflexes were present on the right and absent on the left; the Oppenheim reflex was negative on both sides.

The urine was dark, contained bile, albumin and blood (the patient was menstruating); there were no casts, no signs of phenol or mercuric chlorid.

The spinal fluid was slightly tinted and under somewhat increased pressure, with a positive Nonne test and ten lymphocytes per cubic millimeter. Cultures from the spinal fluid made on broth and blood-agar plates remained sterile after forty-eight hours' incubation. Acid fast stains failed to reveal the presence of tubercle bacilli; neither were other micro-organisms found.

The blood examination showed 50 per cent. hemoglobin; 3,700,000 erythrocytes; 37,000 white cells; coagulation time, twenty-two minutes. The blood Wassermann test was unsuccessful as the blood was hemolyzed.

Temperature on admission was 105.2 F. (rectum), pulse 120, respiration 24. The next day the forenoon temperature was 100; the abdominal tenderness was marked throughout and a friction rub appeared over the pulmonic apexes. The stools showed presence of blood, and the gynecologic examination revealed a soft cervix discharging blood and mucus and admitting two fingers. The blood from the uterus appeared in twenty-four hour cultures sterile. The patient died the next day (March 27, 1920).

Clinical Diagnosis.—The diagnosis was hemolytic jaundice. Necropsy was performed fifteen hours after death by Dr. Fred Stangl, the cadaver having been kept in the refrigerating room. The body was well nourished, the skin intensely yellow and showed a distinct crepitation. The uterus, mediastinal fatty tissue and pericardial sac were "ballooned" with gas; the left pleural cavity and the pericardial sac contained bloody fluid (about 600 c.c. in the former); gas was also present in the left cardiac ventricle, myocardium, vena cava, hepatic veins, spleen, liver, kidney and lungs, which were edematous and showed distinct crepitation.

Brain.—Calvarium could easily be removed; the leptomeninges were opaque and contained bubbles of gas and free fluid which was blood stained; the convolutions were wide and flattened; the sulci narrowed. The brain weighed 1,260 gm., was soft and did not crepitate.

The bacteriologic examination revealed *Bacillus aerogenes capsulatus* isolated in pure culture from the heart's blood, pericardial fluid, cerebrospinal fluid, peritoneal fluid and spleen, all taken from the body at necropsy.

Anatomic Diagnosis.—The conditions found were gas bacillus bacteremia; menstruating uterus, gas bacillus infection of the uterus, hematogenous disseminated gas bacillus infection of the heart, lungs, liver, kidneys, spleen, brain, subcutaneous and muscular tissues ("foamy organs"), serosanguineous bilateral hydrothorax, hydropericardium and hydroperitoneum.

Microscopic Examination.—The brain had been fixed in toto in 10 per cent. formaldehyd solution and cut six days after it was hardened. Frontal sections revealed an enormous amount of cavities (Fig. 1), especially in the region of the large basal ganglions and the cerebral white substance (centrum semiovale, internal and external capsules, corpus callosum), partly in the cerebellum, pons and medulla, while the cortex was macroscopically normal throughout. The cavities of various dimensions and irregularly scattered were divided from each other by septums of various thickness, some as thin as paper, giving the brain the appearance of Swiss cheese or of a crumb of bread. The microscopic examination of celloidin sections also showed much smaller cavities, especially in the gray matter of the brain, causing a sieve-like appearance of the section, "état criblé" (Figure 2).

Aside from the large and small cavities, the microscope revealed a large number of vessels packed with thrombi, which exclusively consisted of gas bacilli. The remaining substance of the brain, scattered among the larger cavities, was in some instances normal, in some broken up and granular, while the pia appeared hyperplastic and the subarachnoid space distended and infiltrated with various pathologic elements.

The foregoing findings—cavities, "état criblé," gaseous bacilli, thrombosis, meningeal and parenchymatous changes—will be taken up in some detail.

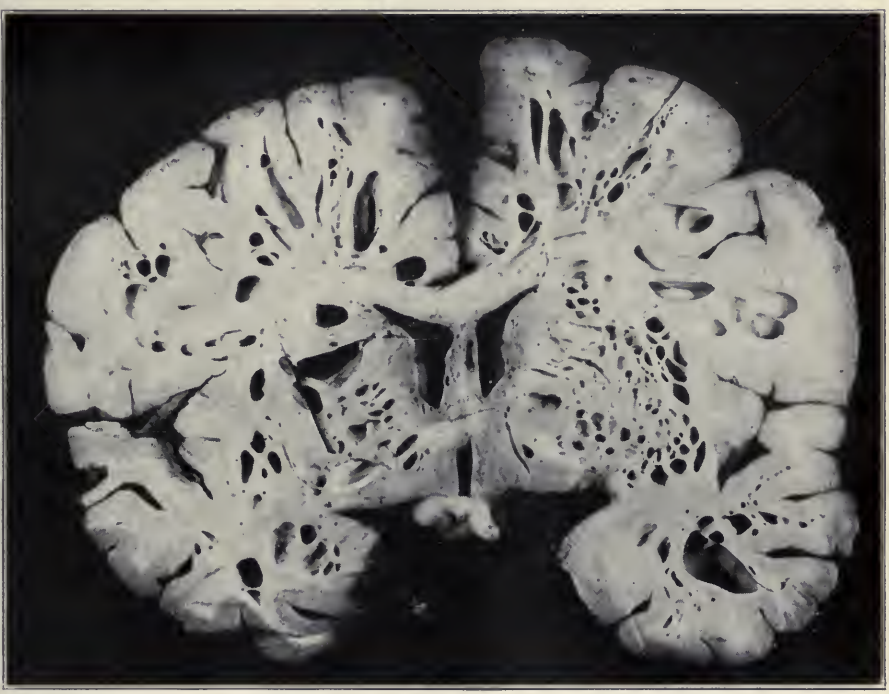

Fig. 1.—Frontal section of the brain through the optic thalamus and lenticular nucleus. The gray matter of the cortex is free from cavities which are exclusively confined to the white substance.

CAVITIES AND "ÉTAT CRIBLÉ"

The majority of the cavities were empty, few contained fragments of broken up nerve tissue (destroyed ganglion cells, fat, globules, débris of nerve fibers and glia tissue), and some distinctly showed the presence of a so-called membrane. Photomicrograph (Fig. 3) shows one type of such a membrane in the form of a broad folded homogeneous band of connective tissue, somewhat irregular in shape and separated from the adjacent parenchyma by a free empty zone. The collagen fibers of the membrane contained oblong sausage-shaped nuclei, rich in chromatin, which were especially well pronounced in loose "membranes," which were represented by thin strings of connective tissue fibers (Fig. 3). "Strings" were unusually common in the cavities, evidently being parts of the adventitial wall of blood vessels detached from the latter during sectioning and left in the cavities.

In the photomicrograph (Fig. 4) a longitudinally cut vessel can be discerned surrounded by a zone of friable tissue which in its turn is surrounded by a condensed stripe of parenchyma forming as it were a line of demarcation. Such a demarcation line frequently surrounds a cavity resembling a distinct membrane.

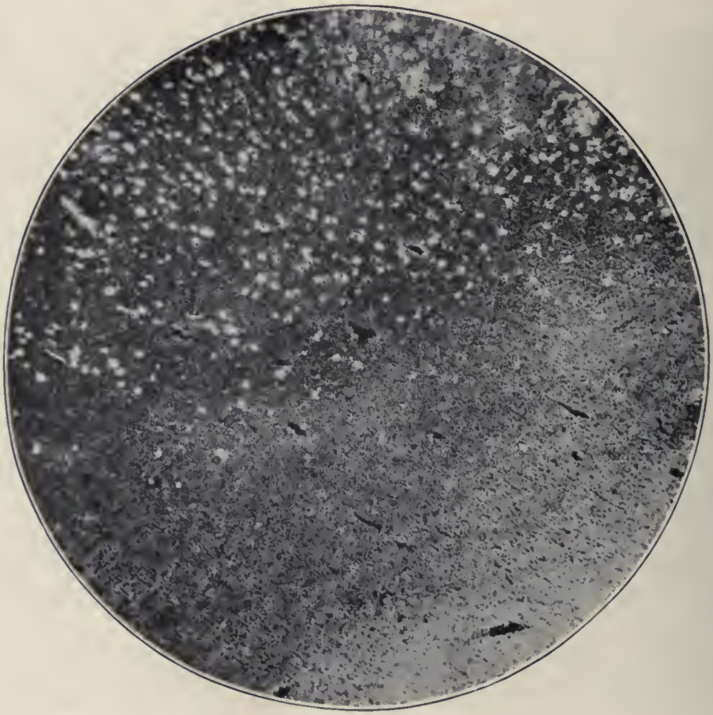

Fig. 2.—The upper half shows état criblé; the lower, with the help of a hand lens, appears granular (the numerous small dots); the larger streaks are vessels filled with gas bacilli. Bielschowsky stain; celloidin section 15 microns thick; × 50.

Other cavities were lined by a dense row of microbes which also may look like a distinct membrane. Large as the cavities might be, they did not invade the gray matter of the cortex from which they were separated by a thickened rim of glia tissue. Only in the gray matter of the large basal ganglions were cavities occasionally found, sometimes attaining quite large dimentions.

As a rule, the cavities in the gray matter were very small, in the form of minute holes the size of a pinhead, giving the section a fine, sieve-like appearance described by Durand Fardel as "état criblé" (Fig. 2). The latter was particularly noticeable in the basal ganglions and less so in the cortex where the "holes" surrounded the ganglion cell and capillaries. There were no membranes in these holes which frequently were represented merely by rarefied glia tissue appearing as a network of pale round minute islands. Both cavities and état criblé were especially in evidence around the vessels which seemed to play a prominent part in their formation. They showed marked and interesting changes throughout the brain.

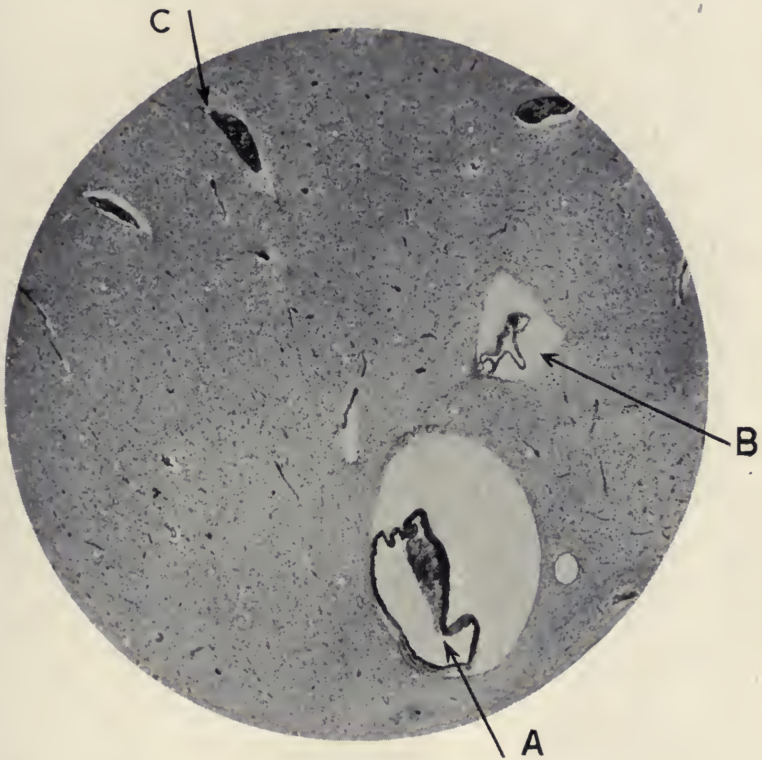

Fig. 3.—Cavities, some of which show remnants of vessels in the form of strings, especially the upper one—*B*. At *A* the vessel surrounds a remnant of brain tissue; at *C* the vessel is surrounded by rarefied glia tissue (this can be seen with a hand lens). Mallory-Jacob stain; × 32.

VESSELS

In some regions, like the cortex, the vessels formed a rich network of capillaries distended and filled with blood (Fig. 5); they showed a well-defined adventitia and well stained endothelial cells, but no dilatation or infiltration of the Virchow-Robin spaces. In some areas of the cerebral cortex and especially in the cerebellum, the midbrain and the pons, there were a great number of smaller or larger veins densely packed with gas bacilli (Figs. 6 and 7), which usually totally obliterated the vascular lumen or preferably covered the vessel walls. The latter sometimes were so densely lined with swarms of bacilli that their condition was hard to ascertain. The adventitia generally appeared more or less thickened but without any pathologic or infiltrating elements and without dilatation of its spaces. Such thrombosed vessels were surrounded by a greatly changed parenchyma which (Fig. 6) was homogeneous, pale, without any nerve fibers or glia nuclei. The few glia cells found in the vicinity were pale, shrunken and usually poor in chromatin. In some instances the parenchyma was distinctly areolar, loosely connected with the blood vessels around which a network of vacuoles was formed (Figs. 3 and 4). The vacuoles, by gradual coalescing, form larger vacuoles or cavities,

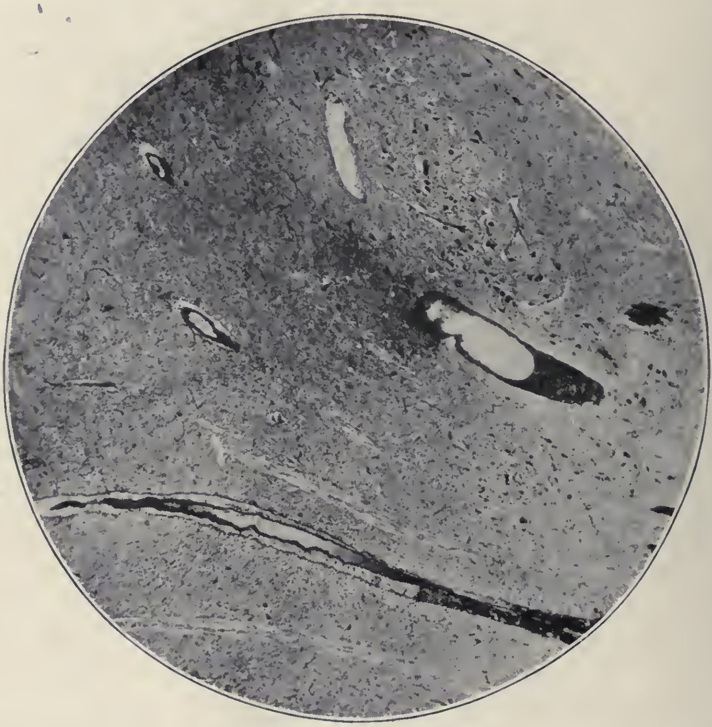

Fig. 4.—Substantia nigra. The longitudinally cut blood vessel in the lower half of the picture is partly surrounded by a zone of rarefied, vacuolated glia tissue which shows distinct demarcation lines especially above the blood vessels. Mallory-Jacob stain; × 32.

or the loose reticular tissue surrounding a vessel may totally break down, leaving a vacant space around the vessel; the latter may even be pushed aside or fall out, leaving a cavity instead. The cavity thus originates around a changed blood vessel (thrombosed) which is the cause of some parenchymatous changes.

PARENCHYMA CHANGES

These were by no means universal. Thus the general architecture of the cortex was intact, the usual array of Brodmann's layers preserved, many ganglion cells showing no abnormalities whatever, but exhibiting marked changes. For instance, in the motor area some ganglions showed a greatly enlarged nucleus surrounded by a free zone, the cell processes being unusually deeply stained and therefore traceable for a great distance; in some the apical dendrones were coarse or broken up, the cell bodies swollen with the nucleus misplaced, somewhat resembling the condition of axonal reaction; other cells were totally homogeneous and invaded by glia cells (neurophagia). Some again exhibited marked chromatolysis. In the caudate nucleus,

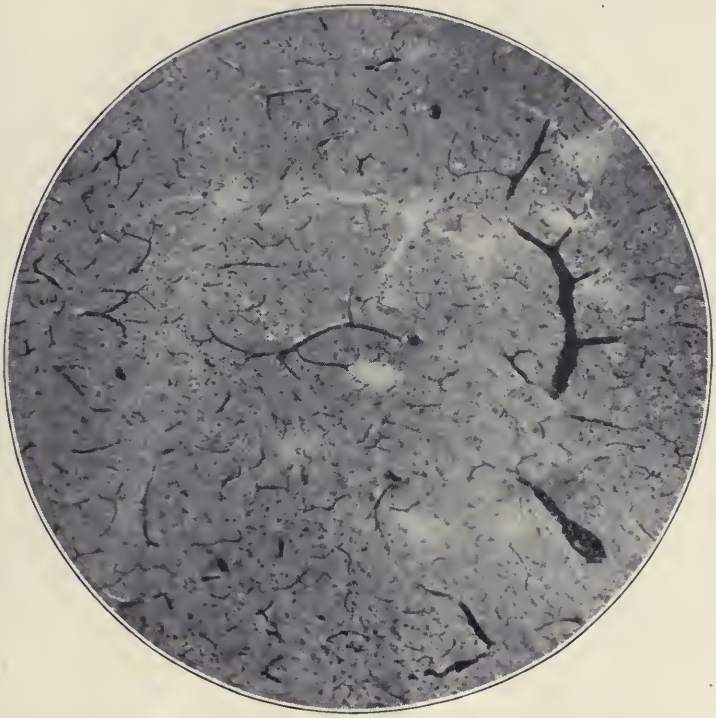

Fig. 5.—Excessive vascularization of the cortex (motor area). Alzheimer-Mann stain; × 50.

substantia nigra and lenticular nucleus, the ganglion cells were mostly pale, their nuclei poor in stainable substance and covered by a twisted or distorted membrane; in the subcortical white substance filled with cavities, the Bielschowsky silver stain (of celloidin sections) revealed in some instances a mass of granular amorphous tissue, so well shown on microphotograph 2. The nerve fibers were coarse, granular, or broken up in globules of various sizes without any reactive phenomena on the part of the glia, which was represented by some dark round nuclei. Herxheimer scarlet red stain revealed no lipoid substance in the broken up areas of the brain tissue, but showed an abundance of the same in the cavities, capillaries and some in the ganglion cells. The capillaries densely covering the cortex were almost without exception packed with fat which in but few instances could be located within the endothelial cells of their intima lining. In the larger vessels, especially those found within the cavities, fat was abundantly represented in the shape of large globules within the adventitial cells while the ganglions contained traces of fat and the glia cells no fat at all.

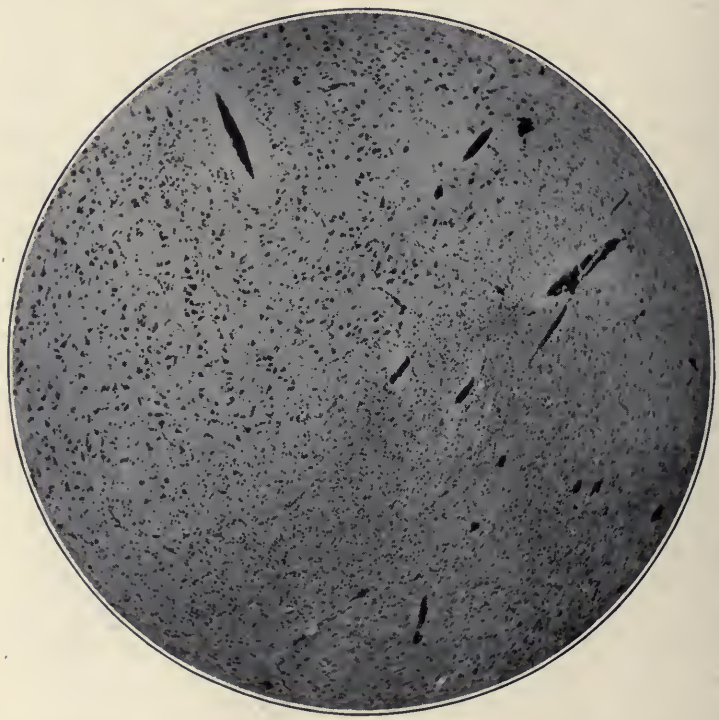

Fig. 6.—The cortical and subcortical regions of a temporal lobe, showing an abundance of blood vessels packed with gas bacilli. The colorless areas surrounding the blood vessels are well marked (use a hand lens). Toluidin blue stain; × 50.

PIA-ARACHNOID CHANGES

Considerably more pronounced were the reactive phenomena in the pia-arachnoid and the subarachnoid space. The pia-arachnoid was hyperplastic (Fig. 8), with greatly distended meshes, usually packed with various cellular elements (Fig. 9): erythrocytes, lymphocytes, plasma cells, polyblasts, macrophages, gitter cells, amorphous granules of hemosyderin and a great number of mesothelial cells (Fig. 10). The deeper layers of pia were densely covered with gas bacilli. In some areas the erythrocytes predominated, forming hemorrhagic foci; in other places the lymphocytes prevailed, mixed with plasma cells, polyblasts and macrophages, while mesothelial cells were abundant all over, frequently forming colonies or, as it were, pure cultures. The fibroblasts sometimes appeared unusually well developed as large oval or round bodies, with few chromatin granules and pale processes. Typical fibroblasts with long and well developed processes were not uncommon. The gas bacilli were either freely scattered among the foregoing elements or were found in large masses within the pial veins, especially around the cerebellum. Even the smallest prolongations of the pia showed in the latter an abundance of blood vessels packed with bacteria. As in the cerebral pia, the cerebellar meningeal vessels were not infiltrated, and did not exhibit enlarged perivascular spaces which were free from any pathologic elements. The pia sub-

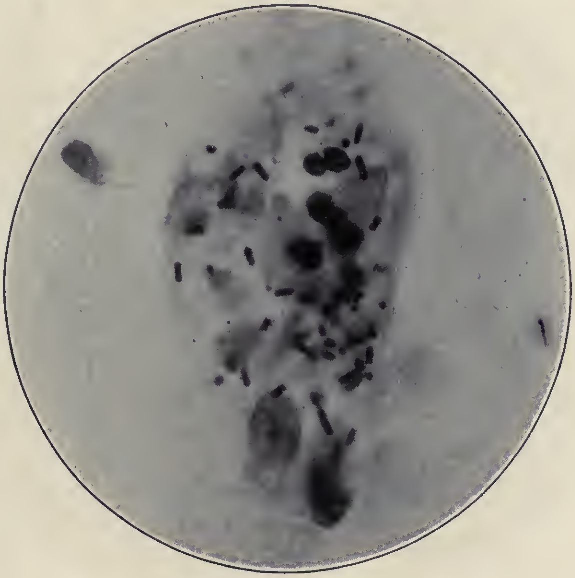

Fig. 7.—Gas bacilli within a vessel. Toluidin blue stain; × 1200.

arachnoid around the pons and lower medulla showed the same wealth of reactive phenomena in the form of the noted cellular elements. In no other pathologic condition could I encounter such a variety and abundance of phagocytic elements, especially of the so-called mesothelial cells—the detached cover cells of the arachnoid membrane and its trabuculae. It is highly possible that some of the phagocytic cells (macrophages, for instance) were transformed mesothelial cells, such a transformation having been so well studied and represented by Essick.[4]

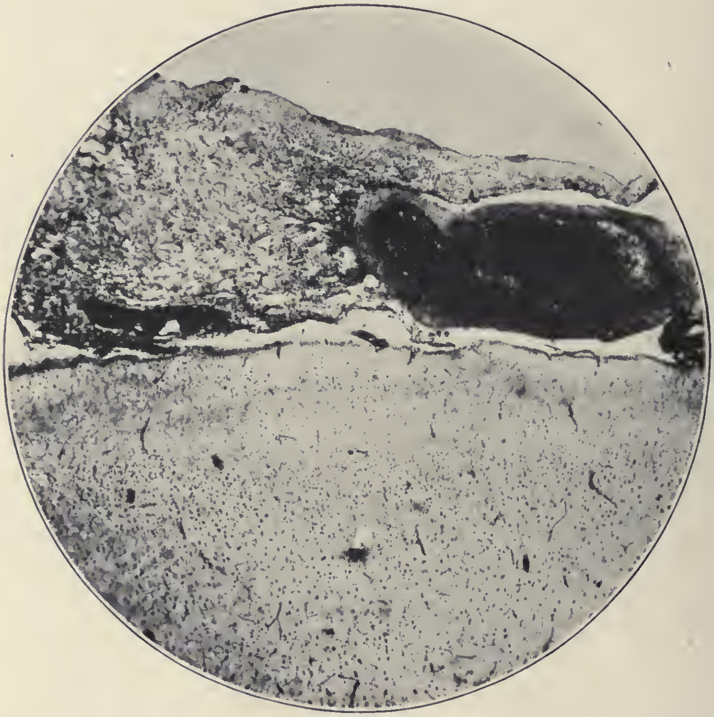

Fig. 8.—The pia arachnoid membrane shows marked hyperplasia, infiltration and a large thrombosed vein; the cortex an abundance of capillaries. Alzheimer-Mann stain; × 321.

DISCUSSION

The most striking pathologic features in this case are the cavities, the bacillar venous thrombosis and the subarachnoid changes. The most striking clinical phenomena are the bronze skin and mental dulness. Both clinical and pathologic findings are most probably a manifestation of the gas bacillus infection, of which the cavities are the most outstanding pathologic features. Repeatedly described and discussed, especially in the older literature, the cavities have been looked on as distended so-called perivascular spaces of His, supposed to be situated between the adventitia of a blood vessel and the brain tissue. The distention is due, according to Ripping,[5] Schlesinger,[6] Pick,[7] Neudoerfer,[8] and others to congestion of the blood vessels associated with increased "lymph" production in the spaces of His. The "lymph" becomes blocked, producing "lateral protrusion" of these spaces and ultimately leading to a cavity formation. Neudoerfer even assumed that the adventitia becomes coalescent with the adjacent brain tissue, thus still more interfering with the discharge of the lymph from the spaces of His. It is needless to dwell further on such views, for the presence of His spaces has not been altogether proved, in fact, is not recognized by the majority of neuropathologists. It would, therefore, be hazardous to build theories on such a weak basis. Moreover, the flow of the so-called brain lymph is not confined to His but to Virchow-Robin's spaces situated between the media and adventitia. These spaces were entirely normal in this case, as well as in another similar case studied by Dinkler.[9]

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[4] Essick, C.: Formation of Macrophages by the Cells Lining the Subarachnoid Cavity in Response to the Stimulus of Particulate Matter, Publication 272 of the Carnegie Institution of Washington, pp. 377 and 388.

Another widely accepted teaching that the cavities are the result of fixation procedures of the brain before it is sectioned, or the result of delayed sectioning (Savage and White)[10] received serious consideration. The fact that the cavities may be found before any fixation has been used and even when the brain is immediately cut after its removal, speaks against such a theory.

The third theory is that of Pierre Marie—cavity formation is probably a product of putrefaction caused by the invasion of gas bacilli. Such a view was also shared by Hartman,[11] who so far has been the only author who could confirm Marie's assumption by demonstrating the gas bacilli invasion. However, he, too, considered the porosis as a cadaveric phenomenon, though he thought that the gas bacilli invaded the body intravitam and proliferated after death. The study of this case, the second in literature in which the presence of gas bacilli could be demonstrated, shows that in cerebral porosis the cavities are not the sole pathologic manifestation, that they are combined with a widespread bacillar thrombosis of the pial and cerebral veins as well as with a breaking up of the glia and brain tissue proper and formation of fat-like substances and marked pia-arachnoid changes. All these changes are evidently premortal, not artefacts; nor are they due to lymph stasis in the spaces of His, but most probably to gas bacillus infection. It may be assumed that the latter mechanically brings about the disintegration of the brain substances, especially around the blood vessels, their severence from the loosened and broken up glia tissue ultimately leading to a cavity formation. The fixation of the brain also may contribute to the porosis, but secondarily by causing excessive shrinking and collapse of the blood vessels as well as of the brain tissues themselves. The latter must

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[5] Ripping: Ueber die cystoide Degeneration der Hirnrinde bei paralytischen Geisteskrannen, Allgm. Ztschr. f. Psychiat. **30**:309, 1874; **33**:424, 1876.

[6] Schlesinger: Ueber cystenförmige Erweiterungen der Hirnrinde, Arch. f. Psychiat. u. Nervenh. **10**:16, 1880.

[7] Pick, A.: Ueber cystöse Degeneration des Gehirns, Arch. f. Psychiat. **21**:910, 1890.

[8] Neudoerfer, R.: Dementia paralytica, état criblé, cyst. Degeneration, Virchow's Arch. **146**:369, 1896.

[9] Dinkler, M.: Ein Beitrag zur Lehre von den feineren Gehirnveränderungen nach Schädeltraumen, Arch. f. Psychiat. **39**:445, 1905.

[10] Savage, G. H., and White, H.: On the Causes of Holes in the Brain, Together with two Cases of Universal Cystic Degeneration, Trans. Pathol. Soc., London, **24**:1, 1883.

[11] Hartman, F.: Eine eigenartige post-mortale Cystenbildung im centralen Nervensystem, Wiener klin. Wchnschr., 1900, p. 963.

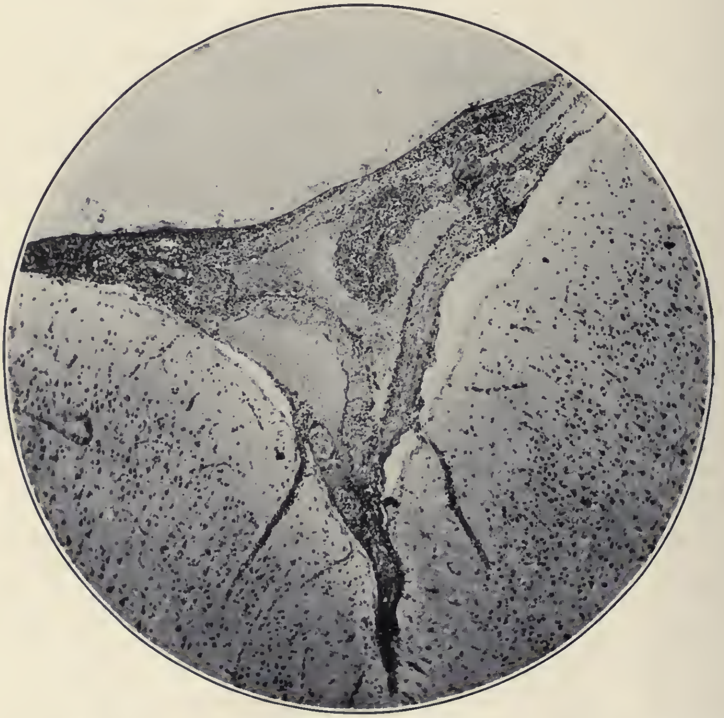

Fig. 9.—Marked infiltration of the pia arachnoid, of the pia prolongations; practically normal appearance of the cortex. Toluidin blue stain; × 50.

be pathologically altered before they can undergo any shrinking; they must first be rendered unstable by some morbid process, in the present case by the gaseous bacilli and excessive accumulation of gas, as the cavities seem to form only in places where the nerve and glia tissues appear friable, and where they are thus easily broken down, either from the direct action of the bacilli or from their mechanical distention and separation from the vessels by the formed gas.

Such a pathogenesis of cerebral porosis seems to conform with the histopathologic findings in this case and seems to justify an assumption that cerebral porosis is a distinct pathologic entity with histopathologic features not to be found in any other infection of the central nervous system. In other words, the brain seems to react in a specific manner against a gas bacillus invasion, just as it specifically reacts against any other infection or intoxication, for instance, paralytic dementia, epidemic (lethargic) encephalitis, lead encephalitis, and other conditions.

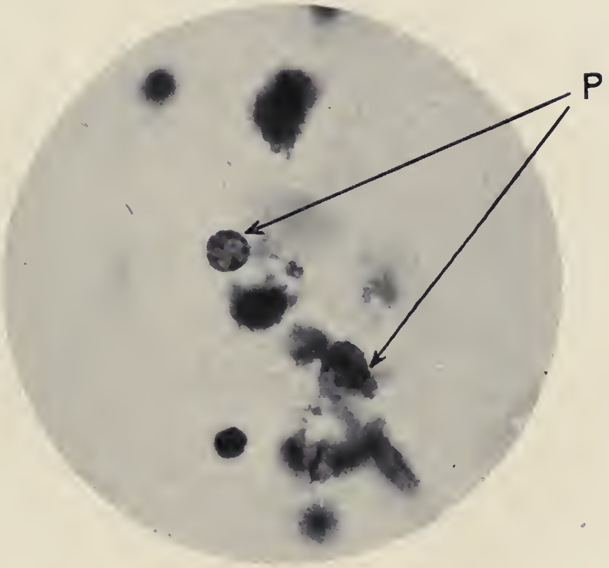

Fig. 10.—Some of the cellular elements of the subarachnoid space reproduced in Fig. 9. Two plasma cells (*P*) and two lymphocytes (dark round bodies) are distinctly seen. Toluidin blue stain; × 1200.

It is obvious that further studies of cerebral porosis cases are exceedingly important and desirable as the existing contributions to this problem, almost all belonging to the older literature, are devoid of bacteriologic and proper histopathologic records, and are not therefore of great value. The previous reports pertain to the most varied conditions. I will but mention the cases of L. Clarke[12] (1870) and Ripping (1874 and 1876), both of general paralysis, the case of Byron Bramwell[13] (1886)—tuberculosis, of Neudoerfer (1896)—a combination of paralytic dementia and tuberculosis, and of Dinkler (1905)—trauma of the skull complicated by dementia. In none of these cases is mention made of the presence of gas bacilli.

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[12] Clarke, L.: A Case of General Paralysis with Examination of the Brain, Medulla Oblongata and Spinal Cord, J. Mental Sc. **15**:499, 1870.

[13] Bramwell, Byron: A Remarkable Case of Disseminated Softening of the Brain with the Formation of Numerous Small Cysts, Edinburgh M. J. **32**:425, 1886.

A SUPPORT IN FACIAL NERVE PARALYSIS

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Facial palsy is rather common and occasionally results in the disability and deformity of complete, permanent paralysis; a less frequent consequence is contracture—but still a deformity. At times these disfigurements may be avoided, yet the condition is too often left to the hazard of "spontaneous cure."

Many feel that in the acute stage of palsy support should be given, and I am positively of this opinion, but the task has been difficult of accomplishment. Several methods have been devised and that recently reported by Jaeger[1] before the New York Neurological Society is said to yield gratifying results. This orthopedist places over the head a net arranged as a cap, to which are attached ribbons and these are fastened at their lower ends to strips of plaster adherent to the sagging cheek. Others have elevated the cheek by means of a hook which engages the angle of the mouth, but this arrangement often causes irritation of the mucous membrane; furthermore, the parts are not brought in proper position.

A device I am using is simple and efficient. Curiously enough, while the scalp at the sides of the head may be moved freely upward and laterally, it may not—fortunately for our purpose—to any extent be pulled downward, and this allows a secure point of attachment toward which the cheek may be drawn.

My method is, first, to cut a strip of plaster 1½ inches wide and about 1¾ inches long, which is firmly pressed well up on the temporal region; this attaches itself securely to the scalp and hair and will remain for a considerable time. Next, a similar strip is cut about 2 inches in length, one end of which is folded on the adhesive side for about a quarter of an inch as reinforcement and into this two perforations are made. Then a strip is cut about 3 inches in length, reinforced as before and correspondingly perforated; the other end is divided longitudinally about 2¼ inches. Finally, two cords are inserted vertically into the perforations and the device is completed.

Apply the support by firmly pressing the smaller strip over the permanent one already adherent to the temporal region, adjust the divided one to the sagging cheek, approximate the free ends and tie the cords securely. The intervening space affords freedom from pressure to the wrinkles necessarily caused by elevation of the cheek.

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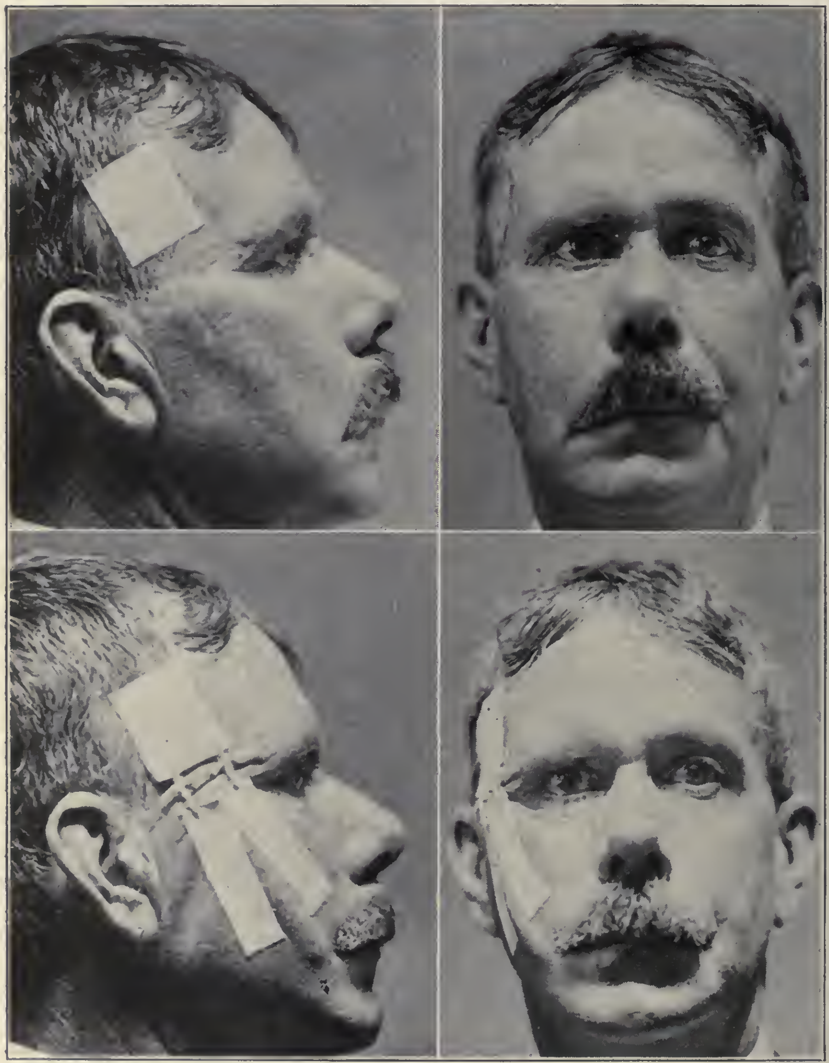
[1] Jaeger: Arch. Neurol. & Psychiat. **1**:374 (March) 1919.



Author's device for support in facial paralysis

This support is adjustable, comfortable, inexpensive, will last for days and, if soiled, may be easily removed and renewed. By no means does the method supplant other forms of treatment, such as physiologic stimulation of muscle action through ideation, internal medication, massage, electricity and diathermia. Briefly, the advantages of such a device are: amelioration of mechanical difficulties such as arise in talking and mastication, hastening of recovery through removal of strain caused by the considerable weight of paralyzed and sometimes edematous tissues on toneless muscles, particularly on the diseased nerve, and also of the blocking of the intermittent tug from muscle action on the sound side. It should prevent some of the disabilities and deformities which one occasionally encounters—hence always fears—in severe facial palsy.

TINEL'S SIGN, FORMICATION OR DISTAL TINGLING ON PERCUSSION AND DEEP PRESSURE SENSATION

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Tinel's sign, formication or distal tingling on percussion (D. P. T.), is, as described by Tinel, a creeping sensation in the cutaneous distribution of an injured nerve when compression or percussion is lightly applied along its course peripheral to the injury. The sign indicates that the axis cylinders are in process of regeneration. The following conclusions relative to the importance of this sign were reached after 2,500 personal examinations in over 500 cases of peripheral nerve injury, operative and nonoperative. The patients had been injured at least three months prior to their entrance to General Hospital No. 6. Examinations were made monthly as a matter of routine.

TECHNIC

With all muscles of the part relaxed, gentle finger percussion is performed, beginning as near the distal distribution as possible and proceeding proximally.

INDICATIONS

1. Progressive formication is not an infallible sign of regeneration in a peripheral nerve. It may be present along the entire course of a nerve, when the greater part of the motor fasciculi are found at operation to be bound up in scar tissue.

Illustrative Case.—S. G. received a double perforating wound in the left arm and forearm Sept. 29, 1918, which resulted in a supracondylar fracture of the humerus, which healed in sixty days. Wrist drop with loss of dorsiflexion of the proximal phalanges and cotton wool and pin prick anesthesia over the dorsum of the wrist developed immediately. Dorsiflexion of the wrist returned about Dec. 25, 1918. Feb. 1, 1919, the triceps, supinator longus, extensor carpi ulnaris and radialis longus contracted normally, but there was no contraction of the other extensor muscles. Formication was present along the course of the radial nerve below the wound in the forearm, but not between the wounds. On exploration of the lower wound, Feb. 18, 1919, the radial nerve was found to be intact, while two thirds of the fibers of the posterior interosseus were divided and the remaining few caught in scar tissue. In this instance tingling accompanying regeneration of the sensory branch would naturally indicate an accompanying regeneration in the motor half of the musculospiral nerve.

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* In charge of Neurosurgery at General Hospital No. 6, Fort McPherson, Georgia, from Jan. 1, 1919, to Jan. 15, 1920.

Following suture of the musculospiral nerve, formication generally advances steadily. We have instances, however, of regeneration of this nerve without formication. Examination of patients with musculospiral lesions shows that the extent of sensory loss bears no relation to the presence or absence of tingling. In one case formication was absent during the entire period of regeneration though the area of anesthesia was typical of musculospiral division.

2. Tingling is not indicative of normal regeneration unless referred to the peripheral distribution of the nerve involved. There is what might be called false formication.

(*a*) A few sensory fasciculi may extend along the trunk for a short distance below the lesion and tingling will be referred to the cutaneous area supplied by these fasciculi.

(*b*) A nerve in close relationship may register a jar. The operator may contuse other nerves in the immediate vicinity and following healing of the wound percussion may cause tingling in an area other than that primarily involved. In several cases of fused neuroma of the ulnar and median nerves, tingling was referred to the distribution of both nerves. In one case of resuture and separation of the lines of anastomosis by a muscular partition, this false type of formication prevailed throughout the period of regeneration. Tapping over the course of the ulnar nerve caused tingling in the distribution of the ulnar and median nerves while tapping over the median caused tingling in the little finger only.

(*c*) A nerve lying close to a bone may give rise to confusing responses. Tapping on an uninjured peroneal or ulnar nerve opposite the head of the fibula or the olecranon will produce tingling not unlike that which accompanies a nerve lesion.

(*d*) Impaired circulation due to interruption of the main artery to the extremity interferes with the proper interpretation of formication. This is especially true in the lower extremity because of passive congestion and edema.

(*e*) Ascending neuritis precludes the possibility of accurate knowledge of regeneration of the nerve involved. The excruciating pain along the entire trunk and peripheral branches, and the extreme nerve and muscle tenderness make examination difficult. In the 550 cases examined there was only one such case.

(*f*) The patient may attempt to deceive. It is essential that the patient's cooperation be obtained. Frequent examinations and careful records will generally detect the malingerer.

3. In a case of regenerating sciatic nerve, percussion over the posterior tibial may cause tingling on the dorsum of the foot. In

such a case the tibial communicating branch of the posterior tibial is responsible, uniting as it does with the fibular communicating branch of the peroneal to form the external saphenous. Unless the examiner is careful this tingling may be referred to the musculocutaneous distribution. The patient should be requested to point to the area.

4. Familiarity with nerve distribution is essential. Where nerves lie in close relationship as do the musculocutaneous, anterior tibial and external saphenous, tapping over the peroneal or musculocutaneous may give rise to tingling on the dorsum of the foot, although the anterior tibial is completely blocked. In testing formication in the leg the anterior tibial should be tested first by gently tapping close to the tibial crest, causing formication over the crest of the dorsum of the foot to the base of the great toe.

5. Peripheral nerve injuries are often associated with cicatrices: Between 40 and 50 per cent. of all patients operated on had scars at the site of the nerve injury which in themselves required surgical attention. Of the patients that came to operation, at least 70 per cent. had no tingling in the course of the injured nerve except when percussion was applied at the site of the scar. The quality of the tingling is of importance in interpreting the underlying pathology. If a patient has only slight or moderate tingling over the entire course of an injured nerve and there is marked formication on percussion over an adherent scar or if the progress of formication has been slow, the condition is sufficiently serious to warrant an exploratory operation, the probability being that most of the fasciculi are divided or compressed in scar tissue. In one instance in a reoperative median nerve case in which formication had advanced at the rate of $\frac{1}{400}$ inch daily, only four fasciculi of a total of approximately thirty were found intact.

6. Formication is of the greatest value in determining the degree of progress after an end to end suture. For several weeks after operation correct interpretation is impossible because tapping near the operative site causes false formication. We believe that formication in a sutured sciatic nerve is of little value until ninety days after operation. In 90 per cent. of the cases percussion within this period over the nerve below the point of suture causes formication either in the distribution of the external and internal plantar or of the external saphenous nerve.[1] The rate of descent of formication varies with (*a*) the nerve, (*b*) the site of suture, (*c*) the degree of tension under

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[1] The tingling in the external saphenous distribution is the only evidence of regeneration in the external cord until the axis cylinder processes reach the head of the fibula.

which the nerve is placed after suture, (*d*) the conditions under which the anastomosis is done, (*e*), the fact whether or not rerooting is done, and (*f*) postoperative treatment.

(*a*) *The Nerve.*—The subjoined table shows the rate of descent of formication in various peripheral nerves following end to end suture:

RATE OF FORMICATION IN PERIPHERAL NERVES

Leg	Fraction of Inch Per Day	Arm	Fraction of Inch Per Day
Sciatic	1/13	Median (arm)	1/18
Peroneal	1/18	Median (forearm)	1/20
Posterior tibial	1/15	Ulnar (arm)	1/17
Musculocutaneous	1/24	Ulnar (forearm)	1/26
Anterior tibial	1/29	Ulnar rerooted	1/38
		Musculospiral	1/18
		Posterior interosseous	1/34

(*b*) *Site of Suture.*—Formication advanced more rapidly after suture in the arm than in the forearm. It advanced more rapidly in a sutured sciatic, less rapidly in the peroneal and still less rapidly in the anterior tibial.

(*c*) *Degree of Tension.*—In some instances in order to overcome a large gap in the nerve, considerable tension was placed on the line of suture; in such cases, progress of formication has been somewhat slower.

(*d*) *Conditions under which Anastomosis Was Done.*—In a large percentage of cases the nerve injury was associated with other conditions, such as bone lesion, ununited fracture, or latent osteomyelitis with sequestrums. In a small percentage a bone operation was done at the time of nerve suture (sequestrectomy or bone graft) and the nerve placed in a muscular tunnel. Formication with one exception advanced normally. This case was an end to end suture of the musculospiral nerve, 5 cm. above the external condyle, with an arthroclysis for fibrosis of the elbow joint, followed by a mild infection involving the periosteum. Reoperation was necessary, and the nerve was found to be compressed and edematous for a distance of 7 cm. Postoperative infections due to the release of an adherent nerve from the bone have not diminished the rate of progress of formication. These cases for the most part were musculospiral sutures. When suture of the musculospiral was accompanied by operation for associated osteomyelitis, the line of suture being protected by a muscular flap, the rate of advance of formication was 1/21 inch.

(*e*) *Rerooting.*—The delay in the advance of formication in ulnar suture in the forearm is in part due to interference with the branches which go to the flexor carpi ulnaris and flexor profundis digitorum. Formication following a rerooting advances more rapidly when these branches have not been stripped back. When a rerooting has been done to overcome a defect in the arm and these fibers have not been interfered with, formication advances to the periphery much more rapidly ($\frac{1}{17}$ inch in a day).

(*f*) *Postoperative Treatment.*—The effect of electricity and massage on the different forms of sensation following suture of a sciatic nerve may be noted in Case 5. So far as we know, only two cases of suture done at U. S. Army General Hospital No. 6 have separated, both musculospiral cases. This was due in the first case to an infection following the insertion of an intramedullary bone peg in an ununited supracondylar fracture of the right humerus, the line of suture being directly opposite the center of the defect. In the other case it was due to carelessness of the ward surgeon, who completely extended the patient's forearm two weeks after operation. The nerve did not separate at the site of the suture, but 1 cm. above. There was no advance in formication in seven months after operation; reoperation revealed the condition. In another patient transferred to this hospital eight months after suture of the external cord of the sciatic nerve in the lower third of the thigh, there was no advance in formication and reoperation disclosed separation and displacement of the distal end to the inner side of the inner cord adhering to it for a distance of 5 cm.

DEEP PRESSURE SENSATION

The following tests were begun with one idea in view, namely, to aid in determining early the presence of regeneration following the suture of a divided nerve. The sciatic nerve was selected because of the large area supplied.

TECHNIC

The foot was kept in warm water for about ten minutes before the reading and the patient placed in a comfortable position. The examiner sat at the end of the table and supported the foot in a flexed position. This is important; otherwise movements of the joint or pulling of the skin may be felt above the area of anesthesia and confuse the patient. Ingham's algesimeter for testing deep pressure may be used, but an ordinary lead pencil with a good rubber tip serves just as well, and since the same person always makes the test, it is not difficult to keep the amount of pressure fairly constant. After repeated trials I find that I use about 1 kg. of weight. Weekly examinations were made. Cooperation of the patient is absolutely necessary; he must differentiate pressure from movement or jarring.

We have considered pressure pain as evidence of return of deep sensibility. We have tried testing with and without blindfolds and our results have been practically the same. Over 500 cases of peripheral nerve injury have been examined during the past year, making in all about 3,500 charts.

Tests following suture of divided sciatics, with one exception, showed that the lighter forms of sensory loss, that is, anesthesia to absorbent cotton and light pin prick, do not shrink week by week or month by month but invariably remain the same even after there is a return of motion. In the area of deep pressure loss, however, there is a definite shrinking which may be noticed as early as four weeks following suture. In some cases, within a period of twenty or thirty days following suture, there may be hyperesthesia of the leg and foot, a development which may be misleading.

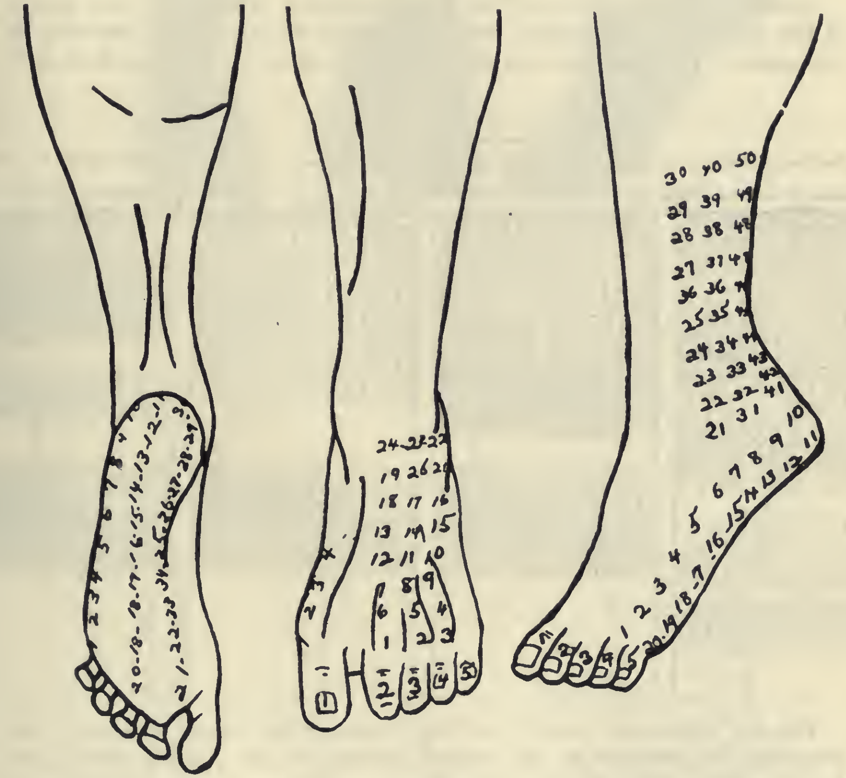
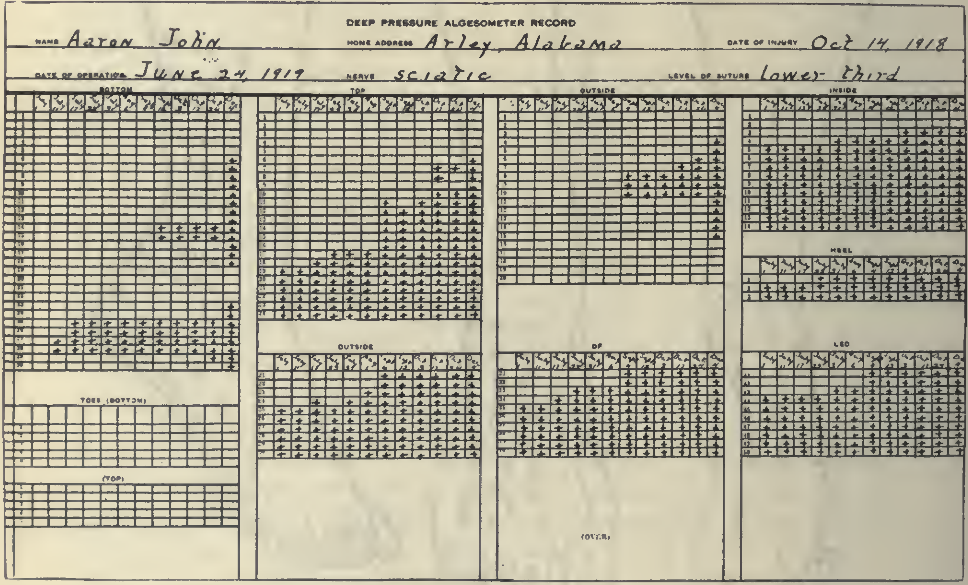


Fig. 1.—Diagram of the plantar and dorsal surfaces of the foot and peroneal surface of the leg, indicating, by numbers, the points for testing the deep pressure sensation.

For recording our results, we have charted the peroneal surface of the leg and plantar and dorsal surfaces of the foot, which are numbered for convenience (Fig. 1). Records of the tests can be kept either on the drawings or, preferably, on the deep pressure algesimeter record sheet; a cross opposite the number corresponding to the one on the drawing indicates that deep sensibility is present at that point (Fig. 2). The reverse side of the algesimeter sheet may be used for records of the operative diagnosis, operation and pathologic report.

REPORT OF CASES

Case 1 (Fig. 2).—Oct. 14, 1918, J. A. received a perforating shell wound of the lower left thigh; there was no fracture, but immediate paralysis of the muscles of the leg and foot and loss of sensation over the peroneal surface of the leg and dorsal and plantar surfaces of the foot; there was no paresthesia or causalgia. The wound was healed June 1, 1919.



DEEP PRESSURE ALGESOMETER RECORD

NAME Aaron John
HOME ADDRESS Arley, Alabama
DATE OF INJURY Oct 14, 1918
DATE OF OPERATION June 24, 1919
NERVE sciatic
LEVEL OF SUTURE lower third

BOTTOM
TOP
OUTSIDE
INSIDE
HEEL
OUTSIDE
OF
LEG
TOES (BOTTOM)
(TOP)
(OVER)

Fig. 2.—Algesimeter record sheet for recording the results of tests of the deep pressure sensation of the peroneal surface of the leg and plantar and dorsal surfaces of the foot.

Examination.—Examination revealed marked atrophy of the leg muscles, a slightly edematous and mottled foot, no fibrosis, dry and scaly skin, thickened and transversely ridged nails, paralysis of the leg and foot muscles; formication extended from the middle of the scar to the plantar surface of the foot and toes. Figure 3 shows the first test, made one week after operation; Figure 4 shows the last reading, five months later. This case shows the typical shrinking of the area of deep pressure loss. The resection and suture of the nerve were perfect; the healing was normal and treatment was begun early.

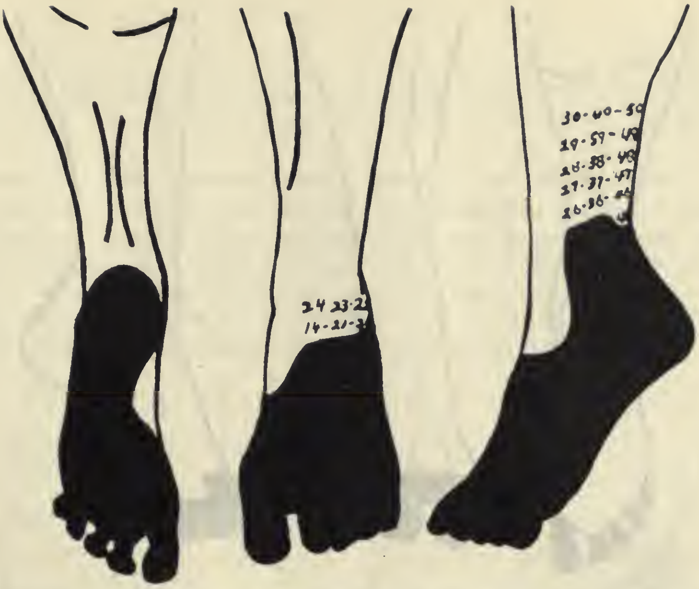


Fig. 3 (Case 1).—Showing the area of deep sensory loss one week after resection and suture of the sciatic nerve.

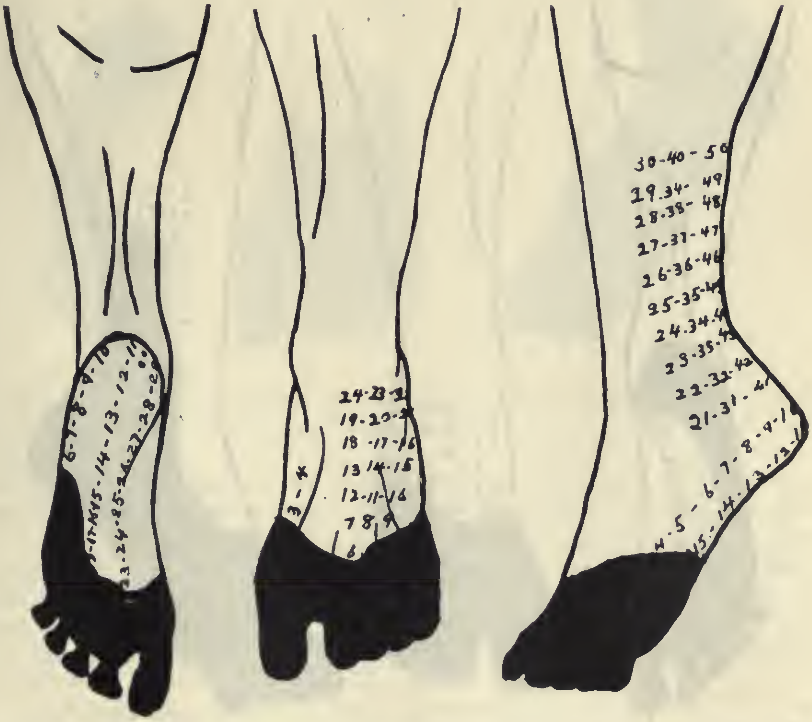


Fig. 4 (Case 1).—Showing the area of deep sensory loss five months after resection and suture of the sciatic nerve.

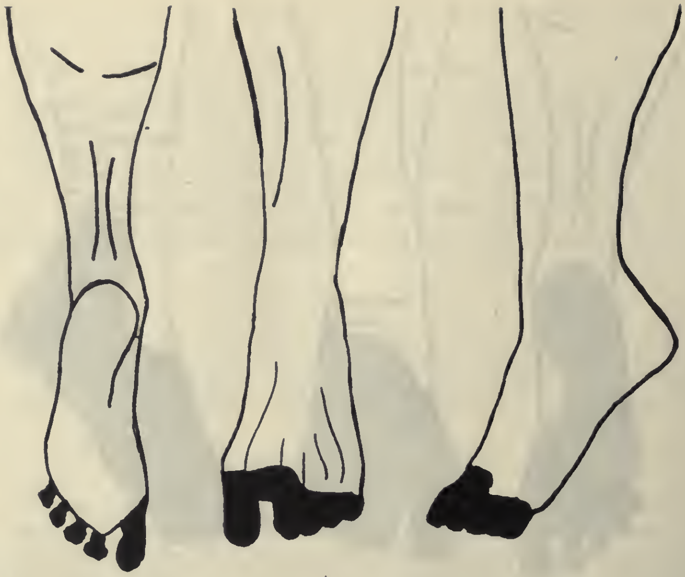


Fig. 5 (Case 2).—Showing the area of deep sensory loss ten months after resection and suture of the sciatic nerve.

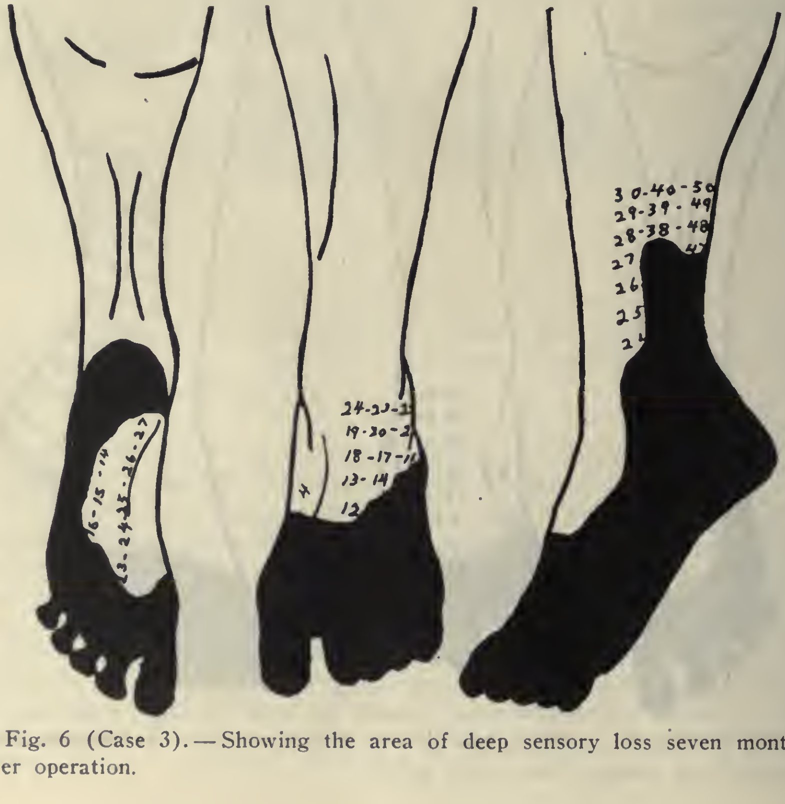


Fig. 6 (Case 3).—Showing the area of deep sensory loss seven months after operation.

CASE 2 (Fig. 5).—On July 4, 1918, J. G. was wounded by a machine-gun bullet which perforated both thighs, in the middle third; there was no fracture, but immediate paralysis of both legs and feet. The wound was healed Nov. 4, 1918.

Examination.—This revealed complete block of both sciatic nerves.

Course and Treatment.—The patient was operated on, neurolysis and fiber dissociation for a distance of 10 cm. being performed on the right side, and on the left, resection of fusiform neuroma with end to end suture. There was complete recovery of paralyzed muscles of the right lower extremity at the end of eight months. Figure 5 shows the area of deep sensory loss in the left foot ten months after operation; there was no improvement over the readings taken five months earlier, indicating some interference with the regeneration. The area of anesthesia to cotton wool and pin prick remained the same.

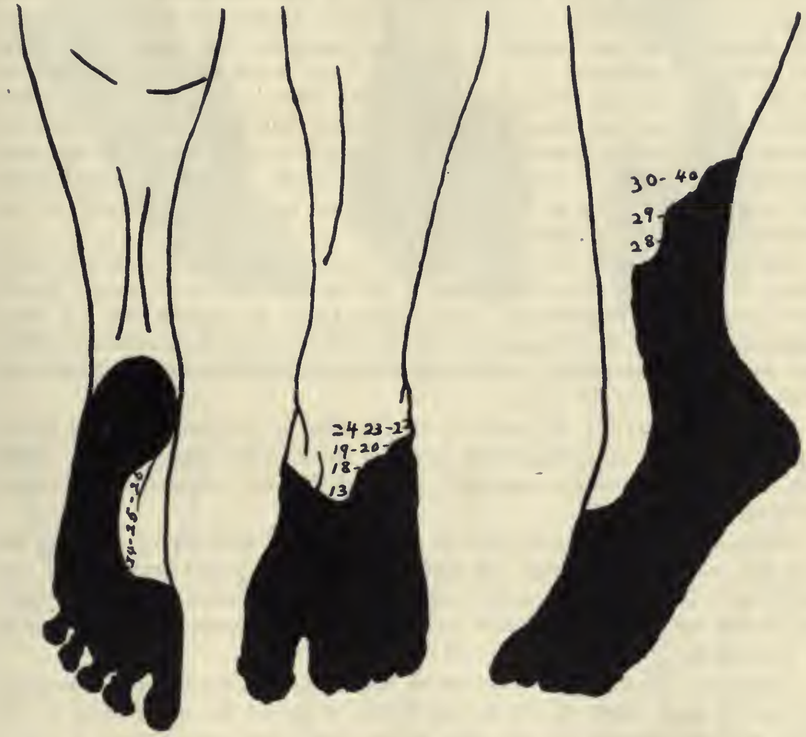


Fig. 7 (Case 4).—Showing the area of deep sensory loss after neurolysis and fiber dissociation of the sciatic nerve.

CASE 3 (Fig. 6).—W. L. received, Oct. 4, 1918, a perforating wound of the upper third of the left thigh, midposterior aspect; there was no fracture, and the wound had healed October 18. He had immediate paralysis of the muscles of the leg and foot, loss of sensation over the peroneal surface of the leg and the plantar and dorsal surfaces of the foot, and suffered intense causalgia of the foot.

Course and Treatment.—Operation was performed Feb. 15, 1919. This consisted of end to end suture, 3 cm. below the tuberosity of the ischium. The chart shows delayed return of deep sensation, the patient having had no massage or electricity for seven months after operation. The circulation of the limb was poor, the skin had a lifeless mottled appearance and the muscles were markedly atrophied. There was improvement in trophic disturbances and deep sensation one month after physiotherapy had been instituted.

Case 4 (Fig. 7).—A. A. M. received, July 22, 1918, a perforating wound of the upper left thigh, with complete block of the sciatic nerve; there was no fracture and the wound had healed, Aug. 9, 1918. Causalgia of the plantar surface of the foot was present for thirty days.

Course and Treatment.—At the operation, Nov. 18, 1918, neurolysis and fiber dissociation were performed; the nerve was not sutured. Following operation, the patient showed no improvement in deep sensation, as shown in Figure 7.[1] At the second operation, Nov. 21, 1919, electroconductive tests showed only fibers to the ham strings intact. The remaining four-fifths of the nerve was divided; a neuroma, 4 by 3 by 1 cm., was found on the proximal end, and the distal end was fibrous and adherent to fibers supplying the hamstrings. End to end suture was done, isolating uninjured branches, and the electroconductive tests following suture showed these branches to be intact. In this case, operation was delayed solely because of apparent descent in formication; later we diagnosed this as a false type, tingling being referred mainly to the internal saphenous distribution.

Figures 8, 9 and 10 show records of deep pressure loss six months after suture of the sciatic nerve, the division having occurred at the upper, middle and lower thirds, respectively. These were taken at random and are quite typical of cases six months after operation. Comparison of these charts shows that the area of anesthesia varies with the level of the lesion, the higher the division, the greater the area.

Case 5 (Fig. 8).—T. B. received, Oct. 8, 1918, a perforating wound of the upper third of the right thigh with complete block of the sciatic nerve. There was no fracture; plantar causalgia persisted for two months. The wound was healed, Jan. 8, 1919.

Examination.—There was marked atrophy of the muscles of the leg; the foot was moderately swollen and mottled, the skin dry and scaly; there was no fibrosis; the nails were curved and thickened; no contraction of the muscles of the leg or foot was made out. Formication extended from the middle of the scar to the plantar surface of the foot.

Course and Treatment.—At operation, March 18, 1919, resection of a neuroma and end to end suture of the divided sciatic nerve of the upper third of the thigh were performed; the branches to the hamstrings were intact.

Case 6 (Fig. 9).—L. P. received, Oct. 23, 1918, a penetrating wound of the middle third of the left thigh, posterior aspect; there was no fracture but complete block of the sciatic nerve. The wound had healed, March 15, 1919.

Examination.—There was marked atrophy of the muscles of the leg; the foot was slightly swollen, not mottled; the nails were normal, there was no fibrosis; there was paralysis of the muscles of the leg and foot. Formication was present from the scar to the plantar surface of the foot and toes.

Course and Treatment.—At operation, resection of the neuroma, and end to end suture of the sciatic nerve of the middle thigh were performed.

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[1] No tests were made as early as this operation. The comparison refers to typical sutures at the same level.

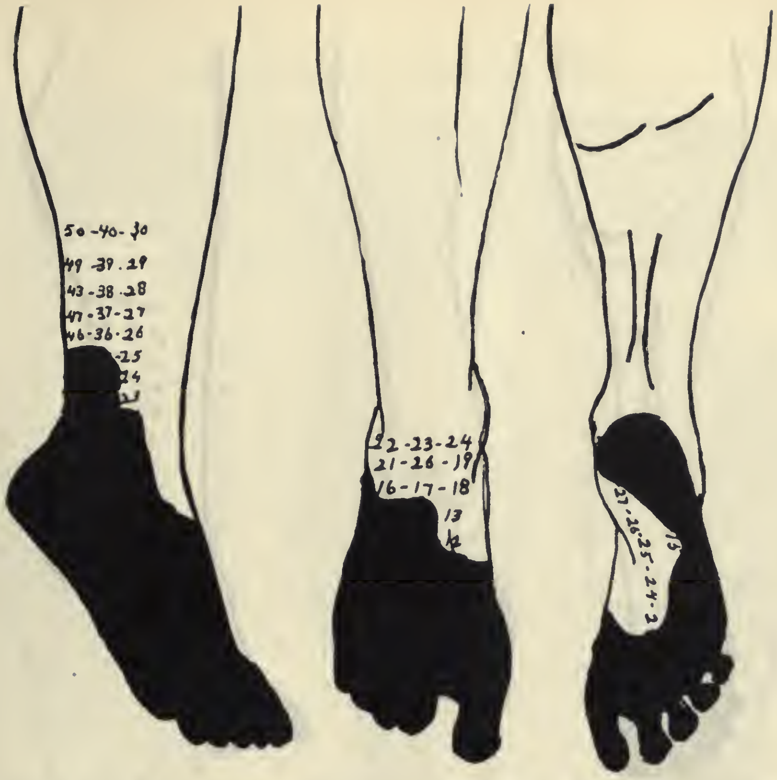

Fig. 8 (Case 5).—Showing the area of deep sensory loss six months after suture of the upper third of the sciatic nerve.

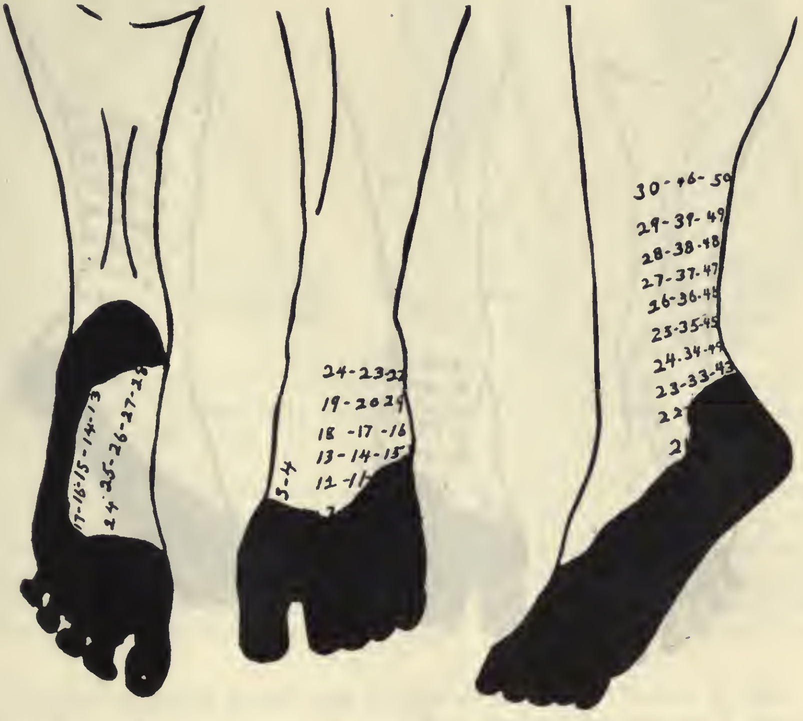

Fig. 9 (Case 6).—Showing the area of deep sensory loss six months after suture of the middle third of the sciatic nerve.

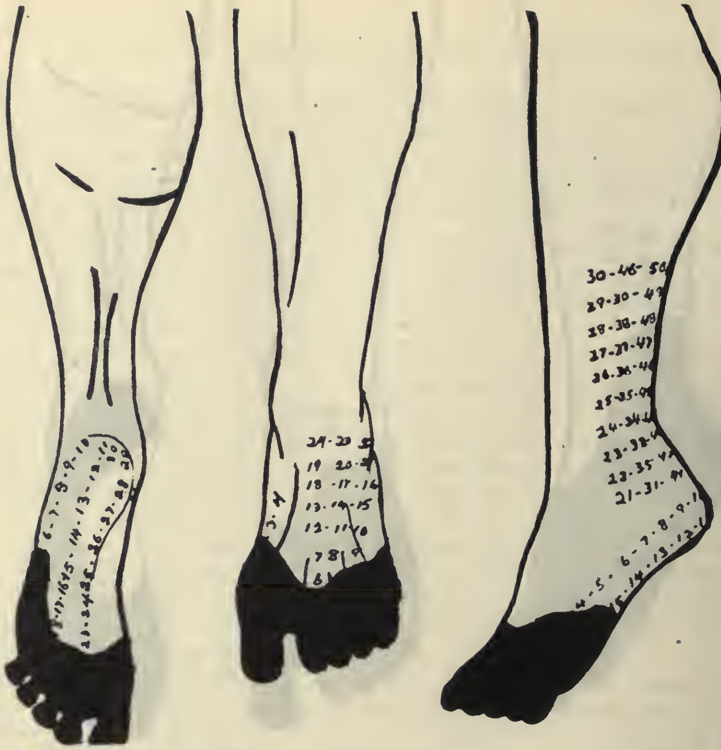

Fig. 10 (Case 1).—Showing the area of deep sensory loss six months after suture of the lower third of the sciatic nerve.

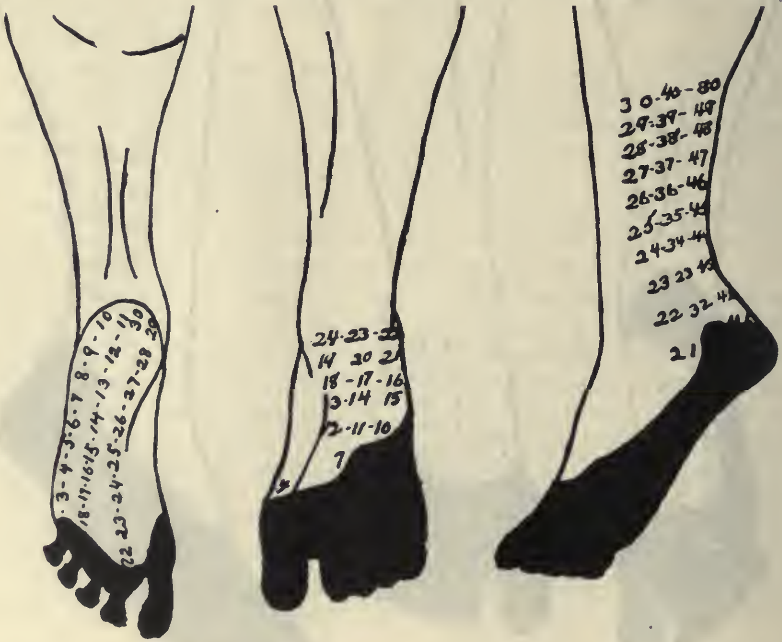

Fig. 11 (Case 7).—Showing the area of deep sensory loss after division and suture of the external popliteal nerve.

Case 7 (Fig. 11).—J. S. received, July 26, 1918, a perforating wound of the lower third of the left thigh; there was no fracture. The wound had healed, Oct. 15, 1918. Causalgia of the dorsum of the foot began two days after injury, and lasted for sixty days. Following injury the patient had paralysis of all muscles of the leg and foot except the gastrocnemius; there was loss of sensation over the peroneal surface of the leg, and the plantar and dorsal surfaces of the foot.

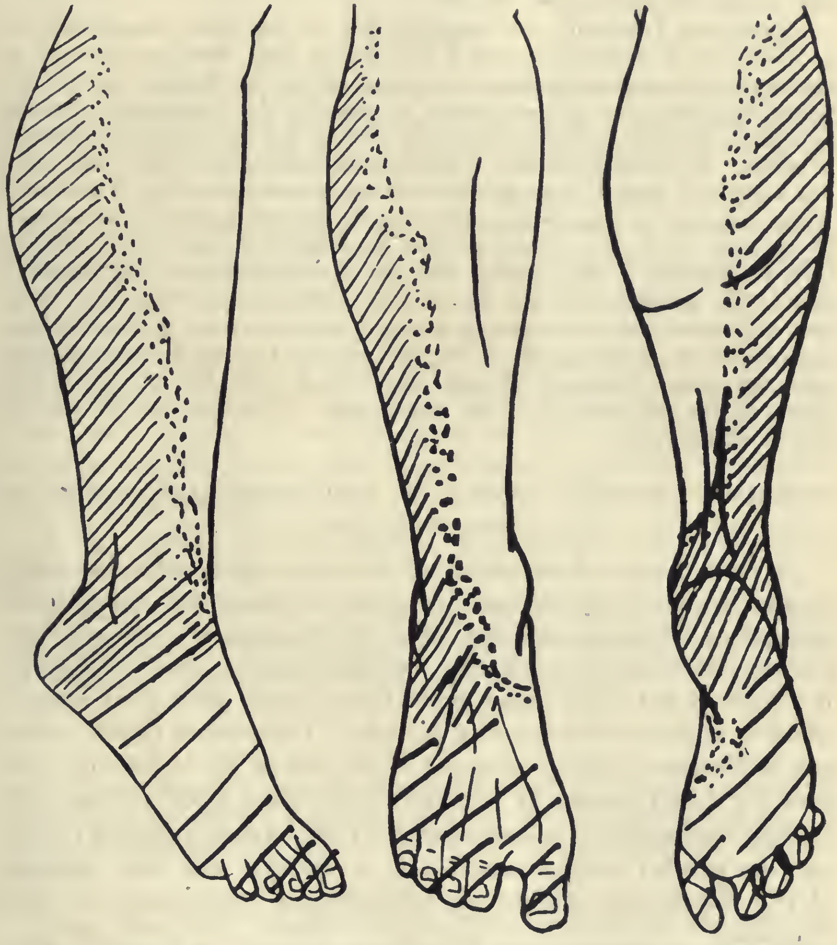

Fig. 12 (Case 8).—Showing light sensory loss with presence of deep pressure sensation in a case of crossing of fasciculi of the internal to the external cord of the sciatic nerve.

Course and Treatment.—At operation, Jan. 29, 1919, end to end suture of the external popliteal nerve and neurolysis of the internal popliteal were performed. Examination, Sept. 11, 1919, showed paralysis of all muscles of the leg and foot except the gastrocnemius. Formication extended over the lower third of the posterior tibial to the external saphenous distribution, and over the peroneal, 15 cm. below the head of the fibula, to the plantar surface of the foot. At reoperation, Sept. 19, 1919, it was found that the external cord had separated at the line of suture, the distal end being displaced to the inner side of the inner cord. End to end suture was done. Figure 11 shows typically the area of deep pressure loss in division of the external popliteal nerve.

Case 8 (Fig. 12).—H. T. received, July 18, 1918, a perforating wound of the middle third of the right thigh; there was no fracture, but complete block of the sciatic nerve. Causalgia over the entire foot lasted for two months. The wound had healed, Nov. 18, 1918.

Course and Treatment.—At operation, Dec. 3, 1918, fiber dissociation was performed for a distance of about 8 cm. During May, there was a return of contraction of the gastrocnemius and during July of the tibialis posticus; following this there was an improvement in sensation and formication continued to advance, but proved to be false in type. There was no further improvement in motion. At reoperation, Dec. 1, 1919, the external cord of the sciatic nerve was completely divided. The proximal end was neuromatous (2 by 1 by 1 cm.). It was necessary to separate the external cord from the popliteal space upward. At the site of the lower limit of fiber dissociation several fasciculi crossed from the internal to the external cord and passed downward; the remaining part of the external cord was composed of fibrous tissue. Figure 12 shows only the lighter forms of sensation absent. Undoubtedly the presence of deep sensation in this case was due to the fact that the fasciculi from the internal cord had grown downward through the external cord; this divergence also accounted for the paralysis of the long flexors of the toes and the intrinsic muscles of the foot.

COMPARISON BETWEEN ADVANCE IN FORMICATION AND RETURN OF DEEP SENSATION

In every instance formication had advanced rapidly when the return of deep sensation was normal; by normal is meant the change which takes place following operation, when the resection has been adequate, healing normal and electrotherapy instituted early. This is illustrated in Figures 3 and 4, the change noted having taken place in six months, which is typical of the majority of cases. Formication in this patient and in Figures 9 and 10 advanced at the rate of $\frac{1}{10}$ inch daily. The tests for deep pressure, as for formication, were made by examiners working independently of one another. Only on comparison of records was the parallel progress noted, i. e., in no case was there progress of true formication without an accompanying improvement in deep sensation.

In Case 2 formication had advanced at the rate of $\frac{1}{40}$ inch a day. This is probably due to incomplete resection or partial avulsion of the nerve following operation.[2]

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[2] Patient J. G., Figure 6, was operated on by the author Jan. 6, 1920. The nerve was divided with the exception of two fasciculi which were nonconductive electrically at the site of the previous suture 10 cm. above the popliteal space. Both ends of the nerve were neuromatous; proximal neuroma 4 x 2 cm., distal 2 x 1.5 cm.; gap between neuromas 10 cm.

In Case 3 there has been no formication since operation, and although the patient received little or no physiotherapy, I am inclined to believe that there is a pathologic condition present to account for lack of improvement.

In Case 4 formication was of the false type, being referred to the internal and external saphenous distribution.

SUMMARY

1. The return of deep sensation is the earliest and most accurate sign of regeneration of the sciatic nerve, the area of anesthesia shrinking in a definite manner and in a definite period of time. This we have not found to be true of the lighter forms of sensation.

2. The term "overlapping" is a misnomer; there is no such thing. When there is shrinking of the area of deep pressure anesthesia, there is without question regeneration of the nerve supplying this area, unless fasciculi which normally supply another area have been diverted from their course by contusion and partial laceration of the nerve trunk. Case 8 illustrates the latter; fibers which under normal circumstances supplied the long flexors of the toes and intrinsic muscles of the foot were diverted by hersage and were responsible for improvement in deep sensation.

3. The psychic effect of repeated examinations is decidedly favorable.

4. There is a definite relation between the return of deep sensation and the advance of formication. When the former returns normally there is an advance in the latter of $\frac{1}{10}$ inch daily.[3]

COMMENT

[With the courteous consent of the authors of the foregoing paper, the Editorial Board requested a comment from Dr. Lewis J. Pollock who has had extensive experience with war wounds of peripheral nerves. His comment follows.]

Deep sensibility is subserved by fibers running mainly with the motor nerves. In a mixed nerve some of the fibers accompany the sensory fibers to the skin. This has been the accepted view for a number of years. Anastomoses between motor nerves is common. Therefore the employment of loss of deep sensibility as a criterion of nerve injury or regeneration is unprofitable except in such a portion of an extremity as is supplied solely by one motor nerve. This condition exists in the leg. But it must be remembered that the internal saphenous nerve may be responsible for some of the pain experienced as the result of excessive pressure on areas adjacent to the skin supply of this nerve and within the borders of the sensory supply of the sciatic nerve.

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[3] The operations on J. A., J. G., W. L., T. B. and L. P. were performed by Lieut.-Col. W. W. Babcock. Pathologic examinations were made by Lieut. Charles H. Manlove. Reoperations were performed by the author.

Overlap of peripheral nerves has been recognized for many years. From the results of both division and irritation of peripheral nerves, it has been shown by Head "that as far as protopathic sensibility is concerned they overlap to an enormous extent." This observation has been confirmed by all subsequent work on skin sensation. The residual sensibility of any nerve is greater in area than the accepted anatomic sensory distribution of that nerve.

In the second paragraph of their summary the authors state: "The term 'overlapping' is a misnomer. When there is shrinking of the area of deep pressure anesthesia, there is without question regeneration of the nerve supplying this area." Inasmuch as this communication deals with deep sensation, it is understood that the foregoing conclusion refers only to this type of sensation. That other investigators recognized the existence of overlapping to deep sensibility may be seen from the insistence of Head and his co-workers that in the examination of cutaneous sensibility, only such methods are acceptable as eliminate the possibility of stimulation of deep sensibility by pressure. Unless this precaution is taken sensation is found to be present in areas anesthetic to tactile stimulation.

If, by this conclusion, the authors mean that after overlapping to deep sensibility had already been established following section of a peripheral nerve, any further shrinking in the area of deep pressure anesthesia indicated regeneration, I would agree with them, with certain limitations.

Unfortunately, there does not appear any illustration of the condition of deep sensibility prior to operation and in only one case, Case 1 (Fig. 4), is there an illustration of the extent of loss of deep sensibility soon after operation, in this instance one week. Here the recorded loss to deep sensibility is far less than the accepted anatomic sensory distribution of the sciatic nerve. If, as in the cases observed by me, the loss of deep sensibility occupies an area much smaller than the recognized sensory supply of a severed nerve, overlapping must occur.

To come to a conclusive decision relative to the overlapping of deep sensibility, it would be necessary to study the loss of deep sensibility when several adjacent nerves are injured, the effect of section of adjacent nerves subsequent to the original injury, and the effect of resection and suture on existing sensibility to deep pressure. In other words, some proof must be adduced to show that there is no overlapping of fibers for deep sensibility. The authors do not include such studies in this communication, and consequently this particular conclusion does not seem to be supported by facts.

Lewis J. Pollock.

NERVOUS SIGNS AND SYMPTOMS AS RELATED TO CERTAIN CAUSATIONS OF CONDUCT DISORDER*

WILLIAM HEALY, M.D.
BOSTON

In no branch of scientific therapy does one need to establish a clearer conception of etiology than in the treatment of disordered or antisocial conduct. The rewards of discovery of causes are here often astonishingly direct.

While developing case studies in one interesting portion of this rich field, which is being more and more opened to medicopsychologists, we frequently discover nervous signs and symptoms significantly correlated with etiologic facts of great moment for interpretation of the case. In stating that such interlocking phenomena are frequently found, we must frankly admit that they are not always present in instances of apparently like causation, and that their discovery does not indicate the same relationships or the same etiology. The lack of standard correlations is, after all, just such as we recognize in the symptomatology of many functional disorders.

It may be best to direct our discussion from the standpoint of some specific type of conduct. From detailed case records we might consider various sorts of misbehavior in which disturbed nervous reactions offered evidence of the etiology. We could take examples of violent temper, sometimes with assaults on property or person, cruelties or definite sadism, various sorts of extreme meannesses, exhibitionism, fetich collecting, deliberate moral contamination of others, to say nothing of other forms of sex misconduct, persistent school truancy, running away from home, desertion, miserliness, malicious mistreatment of property, setting fires. Many of these are quite evidently based on disordered emotions or on the distorted play of instinct, or otherwise rather obviously connote the possibility of accompanying nervous or mental disturbance.

For the best example of misconduct that would seem to be quite unrelated to nervous phenomena of any kind, except possibly those concomitant with fear and shame, we may take stealing; sometimes cases of the usual type of thieving, but in other instances abnormally motivated stealing, thieving not engaged in for the easily understandable purposes of gain or possession. Cases of plainly abnormal stealing are sometimes brought directly to the specialist in nervous and mental disease on account of the unusual character of the conduct motivation, friends naturally thinking of the possibility of some pathologic condition of the nervous system. From our extensive material we can offer sufficient illustrations of nervous reactions related to this single form of misconduct to serve adequately the purposes of our present text.

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*Read at the Forty-Sixth Annual Meeting of the American Neurological Association, New York, June, 1920.

SUBJECTIVE PHENOMENA; REPORT OF CASES

Beginning with subjective phenomena we find in our records numerour instances of headaches, "queer feelings" in the head, feelings of excitement and even dizziness, cases in which these symptoms have been significant clews of etiologic factors.

Case 1.—Take the case of a girl now 15 years old, of somewhat limited mentality and good physique, whom we have had under observation for almost two years, who, up to the time we first saw her had repeatedly stolen in petty ways from homes and sometimes from other places. She herself doubted whether a single month had gone by since she was 5 years old that she had not stolen. She had taken small sums of money and all sorts of trinkets, in spite of corporal punishment and many other efforts at disciplinary treatment by her own respectable family and other people with whom she was placed. She purloined many things of no particular value which she could not use in any way, secreting objects, being generous with money taken, and once unraveling part of a sweater that was being knitted by a member of the family and hiding the yarn. During analysis we found that she derived great excitement from her stealing episodes and a definite sensation of dizziness. Sometimes she wanted to take something, anything, and if she did dizziness and excitement followed, and a little later perhaps a feeling of remorse.

Of course such symptoms indicated some disturbance in her mental life and the revelations of her mental content made the fact particularly clear in this case because the girl herself so plainly worked out for us the links in the chain of associations, going back to early experiences. These involved disturbing sex experiences, first acquaintance with sex words and ideas, together with first knowledge of stealing. Recurrent experiences led to ideation and imagery almost obsessive, which persisted for years. We have her own account of the curious mental mechanisms involved. For example, at 13 years of age, returning for a visit to her home town after a long absence, she engaged in an orgy of stealing foolish things. This was all just after seeing again one of the participants in the original sex-stealing situation, a boy whose name, of course, she had forgotten, but could recall after a time with us. "I took the things only after I saw him." At this time she hid the articles stolen and repeatedly went to look at them, each time experiencing the peculiar feeling which she describes as excitement and dizziness. This is one of the cases in which the analysis of the situation resulted in immediate and continued cessation of the stealing, with no recurrence of the nervous symptoms.

Case 2.—An active-minded, unusually coherent boy of 9 years, given to such excessive stealing that it was practically a daily affair, reiterated to us at several interviews his marked feelings of nervous upset on occasions when certain ideas recurred to him. After recognizing his nervous reactions during mental testing we found nothing but possibly his moderate coffee drinking to account for them. Placed in a good suburban home he continued to steal with such frequency that two months later we were asked to see him again and made a much more thorough inquiry. We received finally a specific account of feelings of great nervousness at times concomitant with certain definite mental content. Through this we were led to a first real understanding of his conduct. It was thoughts of a boy, now dead, who had taught him at the same time sex affairs and stealing. "When I feel like taking things I feel nervous and kind of dizzy, and when I think of those niggers and those girls I think of upsetting the whole world. I get excited. I feel funny all over. I feel like turning the whole house upside down on its chimneys." Exploring these experiences and mental associations seems to have started a new career for this boy in spite of the fact that he earlier remarked, "Now I have got started I never can stop. I have two bad habits and one of them is stealing." During several months now he has not stolen, the first break in a delinquent career that had already lasted a long time. It was not his placing out, it was not merely a new environment that turned the trick; as a matter of fact, he stole daily when placed out after first seen by us and when no prolonged or careful analysis was undertaken. The ordinary physical and mental examination threw no light on causation and accomplished nothing therapeutically.

Case 3.—A boy, 8 years old, of good mental ability, positive and nonsuggestible, who had already been complained of in court by his parents for stealing and repeated running away from home, on study by us was found to be quiet, serious and decidedly reticent. We had to see him over and over to acquire any fair understanding of his background, although we early found that he had times when his "head hurt." It was most significant in this case that, reticent and resistant though this boy was, he voluntarily came again and again to see us. It gradually developed that he had "funny feelings" in his head, "cross and mean" feelings which were directly connected with his thoughts about what bad boys do to each other and about a word, "It begins with f," which a boy had spelled out to him the year before. "He learned me how to spell it. He wrote it on the wall. He swears; he takes money from his mother. He told me about stealing."

Case 4.—Another boy, 9 years old, we saw because he was complained of for serious delinquencies, truancy, unmanageableness at home, as well as stealing. He was a boy of good mental ability and fair physical development with some pathologic conditions, namely, slight otitis media and carious teeth. He complained of headaches. We advised, of course, treatment at the hands of specialists and, because of home and neighborhood circumstances, we advocated his being placed in another home. Here for some months he did well, improving much in his behavior except in regard to stealing, and in physical condition except for headaches. We saw him once more and again found in this case that our previous examination, while including the ordinary inquiries, had not reached far enough into either the possible causation of his headaches or stealing. He now seemed most anxious to fathom his difficulties, and his own statements were most illuminating. He feels "rotten and queer" at times, he tells us, and those times are when what he heard about bad things comes into his mind. It is bad words which make his head ache and they make him dizzy, too. He tries hard to get rid of them. "It makes me think of getting into places and taking things." Just before leaving us he said, "When they come in my mind, I try to cross them out, like this (gesturing an X) and sometimes they stick to me and sometimes they don't."

Case 5.—From the cases in which headaches have been reported we may also cite the instance of a boy of 13 years, good in mental ability and normal in physical development, who demonstrated nervous tendencies by his tense, frowning expression and jerking, incoordinate movements. At home and to us he complained of severe headaches; they were in the back of his head; they came on at night; the boy stated that he used to get out of bed and cry with the pain. His mother put cotton in his ears for it. Worry about words brought on the headaches—bad words in bad stories. The things that had bothered him most in his life had been the bad things he had heard about girls and about stealing and these headaches. He would like a chance to stay in a quiet place, he said.

This has been one of our most unsuccessful cases. Placed with good people, his temperament and the death of both parents seemed to stand in the way of his overcoming his delinquent tendencies. Further stealing led to his commitment to an institution. Before this and several months after we saw him first he reported that his headaches had ceased.

Case 6.—In our Chicago series there was a boy of 8 years, who was considered a desperate problem because he stole. The mother had long known of certain headaches; he complained to her frequently at night about pain in the back of his head and queer feelings there. Pointing to the occipital region he had long ago told her that a word hurt him there. Of course she paid no attention to such a strange remark and had merely told him not to think about it any more; she thought he ought to have some medicine for his headaches. Coming from an open-faced, frank, pleasant, bright-eyed little boy who, in spite of excessive stealing, was reported to be a consistent truth teller, his story to us proved most striking. He went into some detail about his headaches. "It goes right to here in my head and goes all around here and gets me dizzy." "I try to go to sleep and it hurts me. I sleep a little and it starts up again. It hurts me awfully in my head; only at night." We asked him what he thought made him dizzy and headachy. "It's what the kids told me. I roll all around in bed and mamma can't stop me. I think about what they told me, about that stuff, and sometimes it goes right up to my eyes." Other extracts from his statements may be used to give the gist of the situation. "I don't know that word now, I can't remember it. I told my mother it hurt me in my head." "That N. boy talks bad. He says go ahead and steal, steal." He did not want to say those words, he told us with a sigh, because his mother had told him not to say bad words. "It hurts me awfully in my head." Six months later the boy was still in the old environment, was seeing the other boys, was occasionally stealing and somewhat indulging in the masturbation which had begun earlier. However, he said, "Nothing hurts me in my head any more, I'm glad it don't." We advised placing him in some other private home in order that he might have the reeducational opportunity that he needed. This was done and half a year later a marvellous change was reported as having already continued for several months; there had been no stealing whatever and his other bad habits had been discontinued.

The sign or symptom of something abnormal is often phrased as "nervousness" by the relative giving the history or by the young person who is the subject of investigation as a conduct problem. Tenseness of manner or feeling, shown often by nervous habits, particularly biting the nails, irritability in social reactions, undue restlessness or tendency to hyperactivity, are evidently what is generally meant by this nervousness.

Case 7.—One of the worst patients with antisocial behavior we have had to deal with in recent years gave us, when he was in his best mood, a vivid description of his own inner turmoil and its effect on him. When he thought of S. (a companion from whom he learned much that was unfortunate and whose name he could not remember at first) he became nervous. "I think of him and then I get all nervous and I bite my nails." "I don't know what's the matter with me; maybe I could get over it if I went somewhere else. Somehow I can't help it because I am so nervous; even when they get sore I bite them and keep on biting them. When I get nervous I want to steal. I'll be playing a game and will want to think of another game and everything will come up in my mind at once and I'll have to sit down." At another time this healthy looking country boy of 11 years, unusually strong for his age, in altogether a different mood, obviously tense and irritable, told us that he did not love anybody, he hated even himself, he would just as soon be sent to prison or anywhere, and our questions made his head ache. Much might be said about this case, about his arrest at 9 years for burglary and recently for burglary with a revolver in his possession, other stealing, about his irritability at home, unkindness to children and animals, wakefulness at night, complaints of feeling nervous, absolute self-centerment, even though completely gregarious. In the back ground, by way of possible causation, directly and particularly, was his association with a couple of bad companions, who stole and with whom he repeatedly had pervert sex experiences.

Placed in a private family for a few days' observation, this boy proved even more impossible than in his own home; he stole a valuable watch, was up at all hours of the night trying doors, broke up furniture, ran away—and so had at once to be sent away to a reform school without anything more constructive being done for him.

Case 8.—Another case with a better outcome was that of a boy, 13 years old, whose wornout mother asked that he be put away because of repeated stealing, truancy, troublesomeness and low marks in school, and his bad and nervous behavior at home. He was fidgety, restless, irritable, had occasional headaches and dizzy spells and seemed to have something on his mind, she told us. He had irregular habits of eating and coming home; had been biting his nails for a long time. In some ways he showed good traits; he was very nice to younger children, even to babies.

Seen by us, the boy proved to be poorly controlled both physically and mentally. He showed incoordinate movements of hands and body, bit his nails and fingers, seemed much worried and repressed. He cried when he was asked to do mental tests, answered questions rather incoherently, stated that he could not say what he meant, and his attention varied greatly. He told us that he felt so nervous sometimes that he put his fingers in his ears when the cars went around the corner. After inquiry he said that it was what happened under the wharves, watching homosexual affairs between the boys, "That's what got me nervous. One fellow showed me a picture, a photograph. The kids said it came from France. I guess it's that stuff made me nervous. Another kid has a picture I won't look at. It all makes me feel queer." He wanted to go to the country, he pathetically stated, because there perhaps he could forget those things and become more healthy. After this exploration the boy was sent to a relative for the summer and then he returned home. The school reports now for months have been the reverse of his previous record, his scholarship is fair, attendance perfect, conduct good. He is much less nervous and appears happy.

In not a few instances the delinquent young people we have studied have given us an account of "queer feelings" that seem to be definite nervous phenomena to them.

Case 8.—As an example we might take the case of a boy highly supernormal and alert mentally, who showed nothing unusual on the physical side, except somewhat poor color, a rather tired look, slight incoordinate movements of the hands, and some undue restlessness. Although only 10 years old, he had been stealing considerably over a period of four years. This dated back, we found from the boy, to the influence of a companion who taught him stealing and masturbation, the latter having been discontinued under the efforts of a woman with whom his mother boarded him. Concerning his queer feelings, of which he definitely informed Dr. Bronner, he said that they were caused by things he had been told at 6 years of age. "If I could forget the words." "They give me a funny feeling all over. They make me feel bad and if I wouldn't feel bad I think I wouldn't steal." It is over a year now since this boy for the first time revealed his inner mental life in its relation to his sensations and conduct. We lately have a report from his family, who brought him to us originally, that a wonderful change came over him at once and has been maintained. He has not stolen in all this time and he can now be trusted with money and is truthful and takes great pride in his honesty.

Case 9.—The queer feelings, the tenseness and the worry were felt by one boy as a sort of an attack, something coming over him, connected with ideas which arose evidently like obsessions in his mind. This was also a bright boy of 10 years, thoughtful and imaginative, who with his perplexed expression and his high-pitched voice appeared to be decidedly nervous and high strung. He had been stealing for two years in spite of good discipline and often sympathetic treatment. Just before we saw him he had looted his father's strong box at his shop, taking $118 in cash, checks and other papers. The checks, etc., he returned by mail and stayed away from home for a week, a rather desperate adventure for a very small boy.

Again, the boy's own words told the story we are here interested in. Although shortly given, they indicate a little of his really mature approach to his problem. "I get sore lots of times. Because that bothers me, see, about the boys. They tell me about the girls and robbing. They bother me, see, when they tell me about girls. Boys write things on the walls and they spell it out like this. It was on X place when I first heard it. I tried to forget it, but I couldn't. I can't leave go of it. I think and think and then I forget what I went to do. Sometimes I don't know what I'm doing, I'm so bothered. The worst is the bad things they say. That $118, I don't know for what I done it myself." Some months after our examination the father reported with much gratitude that there had been no further trouble with this boy.

As we should naturally expect, the general feeling of irritability may be expressed objectively by exhibitions of crossness or mean behavior to others.

Case 10.—A child of 8 years, who had been stealing much, setting fires dangerously several times, was mean to other children, quarrelsome and showed violent temper spells, said, "The words, I think of them, sometimes I say them to myself. When I think of them I get into mischief. The words make me cross. I think of these words and of bad boys and I do everything I'm not supposed to. I feel mean when I do those bad things. I worry; I want to know what the words are."

Case 10a.—A sullen and evasive boy of 9, called unmanageable at home as well as a little thief, wrote certain words for us and says, "When I think of these I feel like everything, I feel cross."

Case 11.—Another pilfering boy of 11 years, active, restless, quarrelsome, domineering, did poorly under our observation for about a year, when our recommendations for placing him were not carried out. Then on further study of him we succeeded in getting facts that seemed to explain the causative background of his bad behavior, and since then the same observers report a great change for the better in his conduct under the same home circumstances. He blurted out, "Something is the matter with my mind. It's because I always have bad thoughts. They don't make me feel good. I feel cross. It's a word." "What is it?" "That's the trouble, I can't tell, I can't say the word because its a mortal sin. The priest says I should say a prayer and sometimes it helps and sometimes it don't." The boy wrote an Italian bad word, refused to write it in English, but said it begins with "f."

Case 12.—A still more specific example is that of a little girl of 11. About her temptation to steal she said, "I feel as if I was angry with them, some boy and girl, and then when I feel cross or angry I stretch out my hand and take it. I say to myself, 'If Johnny was stealing, I can take it if he could.' I feel angry before I take things, and then afterward I shake like everything." It would take pages to do justice to the mechanisms of this girl's associations as they were slowly developed in her self-revelation. She told how cross she became when boys or men tried to make up to her, she told of her irritations at certain phrases which suggested to her certain other bad things that she had heard boys say. She reiterated that it was her crossness aroused by either kind of experience that made her feel like stealing. The framework of this dynamic associative structure seemed to be as follows: Brought up in a home of great repression, she reacted, with extreme feeling and without confiding in anybody, to exhibitionism on the part of a boy whom we may call Albert. Some time later a second boy, Jimmy, said in her presence some obscene words to which she gave sex meaning. Then and ever since these words or other similar obscenities recalled this affair with Albert. Next in the associative structure was the fact that Johnny, who stole much, as she happened to know, was the chum of Jimmy. Without any other sex experience, the middle element, obscenity, or ideation concerning it, or casual contact with men or boys, or even their friendliness, recalled either or both of the other elements, ideation concerning exhibitionism or stealing. The crossness and anger that were aroused might then be relieved by stealing. "Now I think I can stop stealing," said this little girl a year ago, after a first day of resistance and a second day of unburdening. And she has.

Case 13.—Another little boy, of 8 years, complained of for excessive petty stealing, told us that he was a member of a secret service club and in recounting the activities of that organization of youngsters said that the bad words he heard "Make me feel rotten, I want to fight everybody I meet, my little sister; I feel cross. I go about like crazy. My brains make me steal when I don't want to." The story of his misconduct quite bore out his own description of his inner mental life; at home and elsewhere he seemed uncontrollable. His teachers reported him bright, attractive, likeable and that if let alone he seemed to go into a dream.

CASE 14.—A nervous manifestation of great interest as evidently related to the causation of a really desperately developing criminal career was found in the case of a boy of 13, referred to us by the Boston City Hospital. The boy had engaged in systematic stealing for years; it had been indulged in with periods of intermission since he was 6 years old. At times it had amounted to an orgy of thieving. Placed in an institution for the second time, shortly before we saw him, he had jumped out of a second story window, but was not seriously injured. The signs of his nervousness were appreciated by his parents, the family physician and at the hospital, but no explanation was forthcoming. One doctor had thought offhand that he might be defective and he was sent to Dr. W. E. Fernald, who found that he was not feebleminded.

The story of the boy's anomalous tendencies is long, but I give a short summary. He was quite honest at home, stealing elsewhere in spells. He was naturally very timid, not fond of athletics, and cried quite easily if hurt a little, yet in his stealing he had engaged in bold escapades. He was afraid of the dark, but had repeatedly slept out all night; once his father found him in a dog kennel. Sometimes he was gloomy and depressed though generally cheerful. He was reticent about himself, though talkative about exploits of other boys. He was generous, giving away anything, courteous and polite. He read many books superficially and had some artistic skill in painting and modeling. He had been nervous for years, biting his nails badly and biting his lips at times. He twirled his hair and picked at his hands constantly if his father took him to task. Sometimes at night in bed he shivered so that he shook the bed. Enuresis was present for a short time when he was 6 years old. (Careful inquiry brought out nothing that resembled epileptic attacks.)

He indulged in stealing strangely. He had taken many chances of being caught and being hurt and he had taken many things for which he had no use. For example, when he had a good bicycle of his own he stole several others, without attempting to sell them. He had done such things as breaking into a place to steal something he had seen through the window; he had made away with a package he saw in a carpenter's shop. He had taken money from school children and many things on many occasions.

It was the boy's own account of his behavior and experiences that was of main interest. At one period of our attempt to analyze with him his own career and reactive tendencies he said in response to our query whether he knew why he stole so excessively, "It's because of things I don't like to talk about." Concerning his ideas of stealing he said, "They come usually when I'm not busy, then I have to start jumping round and then I forget about it." Talking of his nervousness he said, "I see something and I shake till I take it." It was like the shaking he had in bed. Several times he told us he wished he could have some medicine that would cure his nerves. He thought he could stop stealing if he could get medicine so that he wouldn't shake. "It would keep me from planning these things. (When?) In the day time or at night. I mean if I am walking along and see something. Then something comes up. I have a nervous feeling and go and take it. I feel nervous all over. I would be looking all over and I would stand still and I don't know whether to take it or leave it there." In response to questions, he told us his heart beat hard then, he shook or shivered, and also had erections. And all this, which may be in front of a shop window, is the same sort of affair that goes on at night in bed. "And when I have that feeling in the toilet I get nervous too." And then he feels "like going out and stealing."

The shaking spells which were made so much of by the boy and his fairly intelligent father seemed most peculiar adjuncts to the situation. At the times when he has these spells he has in mind or has just had in mind, so he worked out the situation with us, a couple of boys who first taught him stealing. But even more vividly than his first experience in stealing with them at 5 years, which his father corroborated, for they were notorious youngsters, stood out in his mind his experiences with them when at about the same time on a couple of occasions in an old shed in a field they undressed him and initiated him in sex practices. They took off all their clothes together there. We hesitated much to suggest by direct questioning and he did not offer any statements of being cold and shivering on that occasion, which seems more than likely. He said that he was not hurt or frightened. He was not sure what time of year it was, but we have some intimation in the fact that it was about this time that he stole with them the first time, and the object was a snowshovel. It seems highly probable that his ideation concerning these other boys and the event in the shed in the field is also associated with subconscious memories of shivering, and the shivering recurs as well as thoughts about the boys.

At any rate, getting this whole affair clearly set forth and faced six months ago resulted immediately in complete cessation of stealing and of the shivering attacks. The father says, "He seems a changed boy."

Case 15.—Peculiar dizziness and nausea were part of the reactions of Tom, a supernormal nail-biting boy, 9 years old, to a situation which involved his brother. Dan, this brother, is a couple of years older, also a bright boy, and we had the opportunity of studying him also and corroborating the facts. Dan had a friend Jim and Jim taught Dan to steal long before Tom knew about it. Dan for a time stole much. Jim also taught him bad words which he passed on to Tom. The latter said he wouldn't say them, he didn't ever say them, he thought them and they made him dizzy. He had never told anybody about them; they made him feel queer; they made him feel sick; they made him steal. When Dan was at the table the words came into his mind and made him sick at the stomach and he couldn't eat. He left the table sometimes.

There is much in the record of this case, too, but in short it may be said that the situation at home was so acute that we advised that this boy be placed elsewhere after this exploration. He did very well, even though his statements to us from time to time evidenced that his brother was much on his mind. We appealed to Dan concerning the use of bad words and then Tom went home and has done wonderfully well for a long time. No more stealing, no nausea is reported. He does not bite his nails so much.

The stuation is especially interesting because of the different reactions in the two boys. Dan had stopped stealing before we knew him; he did not repress the words; he did not worry about them; he was altogether an objective little chap; he had no mental conflict.

OTHER PHENOMENA ASSOCIATED WITH CONDUCT DISORDER

There are other phenomena that deserve at least some mention: Occasionally tics must have significance for us as not fortuitously occurring in connection with delinquency, as in the instance in which a boy said that not only his impulses to steal but also his twitching of the face and half-shrug of the shoulders followed a miserable experience with an older delinquent. He said, exhibiting his tic, "Its when I think of him that I do like this." Or it certainly indicates something when a wholesome-looking little girl, who repeatedly steals even directly after punishment, persistently looking at the ceiling when questioned, not answering although compressing and moving her lips, furiously wrings her hands. The latter action, indeed, had been rightly recognized by teachers and others as a sign of nervous or mental disturbance in her case.

A boy, reported nervous, says that he is so nervous that he shakes or gets hot and sweaty and has intestinal symptoms as he thinks of stealing and of the circumstances that surrounded his earliest acquaintance with stealing. The nervous symptoms peculiarly disappear in the act of stealing; there is no excitement and no fear afterward, as one would naturally expect. He is another boy that pleads for a chance for a quiet life.

Stuttering is so genetically related to character abnormalities in certain cases of delinquency that it has received special recognition by neurologists and psychologists. And the same is true of hysteria—we need not dwell on the protean manifestations of this condition as connected with delinquent careers.

IMPORTANCE OF MENTAL CONFLICT IN CONDUCT DISORDER

There is much that is of incidental interest in this whole discussion; for example, the peculiar perplexity and concern of these young people about their own mental affairs, the fact that so many of them are far above the average in mental ability, their frequent forgetfulness of the names of persons whose memory is bound up with their earliest experience with delinquency and unpleasant feelings, their great resistance to sex words, being willing, however, to write them or say the first letter, their own reaction to their own story—some have asked to be allowed to continue at another time because there is more to tell—the strong signs of repression with many, the emphasis which they place on their own feelings of queerness, so unusual in children. Only rarely have dreams seemed to be of value to us in getting at essential causations.

Thus, in connection even with a form of delinquency in which we should least expect it, we find many nervous phenomena—feelings of worry, of being bothered, of depression, of tenseness and general nervousness and excitement. And more definite symptoms—dizziness, headaches, nausea, nervous exhaustion—are reported. Plainly to be observed are biting of the nails, pulling out of hair, marked restlessness, shaking and shivering attacks. During analysis of the case all these are clearly stated by the subject to be specifically related to certain ideation recognized as undesirable. Perhaps there is nothing any more remarkable in this than that physical or specifically nervous signs and sensations accompany other ideation with emotional context, events in sex life, thoughts accompanied by anger, fear, embarrassment, etc. At any rate it comes about that recognition of the significance of these nervous signs leads, in not a few instances of delinquency, to unearthing etiologic facts and experiences that are of prime importance for making over the individual, nervously and morally.

I have set forth elsewhere that just such a background as is depicted in the cases cited may lead to a prolonged career of misconduct, even of criminality. The establishment of habit, of a set of mind goes on here as elsewhere. Perhaps the nervous symptoms tend to decrease or cease during the course of years, while delinquencies persist, but we have seen numerous instances of their continuance well into adolescence and sometimes to young adult life. There is no escaping the fact that this type of a career, neurotic and delinquent, is a matter of grave concern, not only to the individual and the immediate family, but to society in general.

Intensive study of a large number of young delinquents has led to the conclusion that about 10 per cent. are sufferers from the inner turmoil that we call mental conflict. It is to the importance of this field and the remarkable possibilities of therapeutic accomplishment that I wish to call attention by stressing cases in which the essential disturbance for some reason has the above outward manifestations or indicators.

News and Comment

ASSOCIATION FOR RESEARCH IN NERVOUS AND MENTAL DISEASES

The Association for Research in Nervous and Mental Diseases was conceived more than a year ago and organized during the following months. Its object is indicated by the title, and it is to be national in scope. The present plan is to have one meeting a year, the subject to be discussed and the contributors being chosen by the program committee. The scientific proceedings are conducted by a commission, members of which may ask questions and require an author to defend his thesis. The commission finally assembles, edits, if necessary comments on, the communications, and publishes all in a volume which is sent to every member. The undertaking is a distinct step forward in American neurology.

The present officers are: president, Dr. Walter Timme, New York; vice president, Dr. E. W. Taylor, Boston; secretary-treasurer, Dr. Foster Kennedy, New York; members of the commission, Drs. Walter Timme, Pearce Bailey, Charles L. Dana, Bernard Sachs and Frederick Tilney of New York; E. W. Taylor of Boston and T. H. Weisenburg of Philadelphia. There is a program committee of eleven members.

The first meeting will be held at the New York Academy of Medicine, December 28 and 29, 1920, at 9:15 a. m.

PROGRAM

Presidential Address, Dr. Walter Timme.
Etiology and Epidemiology, with Especial Reference to the Alleged Relationship Between Influenza and Lethargic Encephalitis, Dr. George A. Soper.
Serology, Dr. Walter M. Kraus and Dr. Irving H. Pardee.
Pathogenesis and Experimental Pathology, Dr. Harold L. Amoss, Dr. Israel Strauss and Dr. Leo Loewe, Dr. William Thalheimer.
Morbid Anatomy:
    Microscopic and Naked-Eye Appearances of the Nervous System in Lethargic Encephalitis, Dr. William G. Spiller, Dr. James B. Ayer.
    Morbid Anatomy Outside the Nervous System, Dr. William Boyd.
    Brain Lesions from Inorganic Poisons Like Lead, Dr. G. B. Hassin.
Clinical Studies:
    Prodromes, Dr. I. Abrahamson.
    Paralysis Agitans and Thalamic Types, Dr. J. Ramsay Hunt.
    Cerebral Types, Dr. J. W. McConnell.
    Cerebellar Types, ——— ———.
    Spinal Types, Dr. Henry Alsop Riley.
    Radicular and Peripheral Types, Dr. Foster Kennedy.
    Treatment, Dr. Edwin G. Zabriskie.
Prevention:
    Immunology, Dr. Harold L. Amoss.
    Psychotic Types, Dr. George H. Kirby and Dr. Thomas K. Davis, Dr. Menas S. Gregory.
Studies on the Question of the Relation Between Poliomyelitis and Encephalitis, Dr. William B. Cadwalader, Dr. I. Neustaedter.
Diagnostic Criteria, Dr. Lewellys F. Barker.
Prognosis, ——— ———.

Additions to Physiologic Knowledge Through Syndromes of Lethargic Encephalitis, Dr. Frank H. Pike.
Ocular Manifestations, Dr. Ward A. Holden.
Vestibular Manifestations, Dr. Isaac Friesner.
Statistical, Dr. I. S. Wechsler.
Bibliography, ——— ———.
Encephalitis Among the Feebleminded, Dr. Karl Menninger.

For information regarding membership, the program or other matters, communicate with Dr. Foster Kennedy, 20 West Fiftieth Street, New York.

Abstracts from Current Literature

APHASIA AND KINDRED DISORDERS OF SPEECH. HENRY HEAD, Brain **43**:87, 1920.

Head, in the Linacre lecture for 1920, comes to certain conclusions as a result of his studies of soldiers injured during the war. The material consisted chiefly, though not entirely, of young men, many of them intelligent, well educated officers. Nothing is said of the pathologic and anatomic investigations which were doubtless thorough and extensive, but reports concerning which are reserved for other communications. We know nothing of the lesions in these cases except that they occurred in one half of the brain, which half is not mentioned nor is it mentioned whether the patients studied are right or left handed. The study is clinical and psychologic and the methods employed in investigating the cases are reported in detail. The author draws a close analogy between his studies of cortical sensations and the findings in his cases of aphasia. He looks on the results in aphasic states in the same light as the partial disturbances resulting from injury to the cortical sensory area. Destructive lesions of the sensory cortex, the author points out, do not result in complete sensory loss of discriminative cortical types of sensibility but rather in irregularly distributed and partial losses. This is true also in hemilateral injury to the cortex of the brain. The speech mechanism is not lost entirely but partially and the disturbance is made manifest in certain fairly definite ways which he classifies under the terms (1) verbal defects (old term, motor aphasia), (2) nominal defects (old term, verbal amnesia or sensory aphasia, paraphasia), (3) syntactical defects (old term, jargon aphasia, paraphasia), (4) semantic defects (old term, verbal amnesia, word deafness, word blindness, alexia, paralexia, agraphia, paragraphia).

Head strikes a tremendous blow at present day conceptions of aphasia and speech development when he reiterates the dictum that there is no memory for words, apart from the thing remembered. He says there are no images of words and no memories of words heard, seen, spoken or written; and speech disturbances cannot be discussed in terms of motor aphasia, alexia or agraphia. The so-called motor aphasic patient has a disturbance of speech in its broadest sense, and careful examination, according to Head's method, shows invariably that we are dealing with a disturbance in some part of all the elements of speech in every case of aphasia. It is not a pure anarthria, as Marie claims, nor a general lowering of special intelligence as it applies to speech (Marie's and Wernicke's sensory aphasia), but all true aphasias show a disturbance of the speech mechanism in its broadest sense.

The author insists that cortical stimuli produce inconstant responses; therefore he emphasizes the necessity of repeatedly applying the tests mentioned below to bring out the speech defect. The author's tests are described in detail in pages 90 to 106. Some are original. The tests are: (1) the naming and recognition of common objects; (2) naming and recognition of colors; (3) the man, cat and dog test. These words of three letters are employed in various combinations, and then the patient is asked to write and read the various combinations and to repeat them after the observer; (4) the clock tests—setting the hands according to command, by imitation, etc.; (5) the coin bowl test—placing coins in a bowl according to written or spoken command, etc.; (6) the hand, eye and ear test—the patient obeys the spoken or written command, or imitates the observer, being seated in front of him, or imitates reflected movements, the observer and patient facing a mirror.

This test is most important, according to Head, because he noted frequently, in the patients tested, a want of appreciation of crossed movement. This is caused by the necessity to repeat the words of the spoken, written or motioned command in order to imitate the observer correctly. Besides these tests, the author had his patients repeat the alphabet or read aloud a story and repeat its contents, read from dictation, etc.

The author disagrees with previous explanations of aphasia. He thinks that "Gross injury confined to one cerebral hemisphere can disturb the power of speaking, reading and writing, without producing any other severe loss of intellectual capacity." A group of functions exist which are "Indispensable for language . . . but not essential for all intellectual performance." The author looks with extreme doubt on reported cases of pure "alexia," "agraphia," and "aphasia." These terms pass out of existence with the passing of the "faculty" doctrine. Inability to speak, read or write, is always associated with some other loss of the speech function. These disorders are not due to the destruction of "images." The images are not lost but the use of the words (symbols) are lost in speech (symbolic expression).

The differences between various forms of disordered speech are due to the breaking up of one aspect of symbolic expression and thinking analogous to the sensory dissociations which follow lesions of the post central cortex. The residual defect after neural shock passes away is the type of dissociation existing in the individual case. Some aspects of speech are more affected than others in these various types, and Head has chosen a name for each of these to express the grammatical error characteristic of each type. He admits, however, that the disturbed functions extend "Beyond the limits of verbalization." However, one can summarize the defects by saying that in "verbal aphasia, words are evoked with difficulty and tend to be abnormal in structure. Nominal aphasia leads to a defective use and understanding of words as names or indicators. Loss of syntactical power disturbs the internal balance of a word as an orderly rhythmic expression and so leads to jargon; whilst semantic disorders interfere with the capacity to comprehend and retain the general significance of a word as part of a complete act of language."

Nothing positively helpful to an understanding of this difficult and interesting subject has been brought out in this article. The author promises further elaboration of the subject in subsequent more important, anatomic, pathologic, psychologic and clinical reports.

Osnato, New York.

THE CLINICAL SIGNS OF NERVE INJURY AND REGENERATION.

Lewis J. Pollock, Surg., Gynec., & Obst. **30**:472 (May) 1920.

The author states that clinical signs of nerve regeneration are manifestations of the spontaneous recovery of an injured nerve, the recovery consequent to the suture of the ends of a divided nerve, or recovery following some operation on an injured nerve which has shown signs of physiologic interruption. To a large degree, signs of regeneration consist in the evidence of return of function.

Many attempts have been made to discover a sign or group of signs which would justify a rapid differential diagnosis between a patient with a complete section of a nerve and one in whom complete loss of function is not the result of division of the nerve. The syndrome of compression, irritation, etc., adopted by Mme. Déjerin and J. Morizon have been proved by others to be artificial and unreliable for the purpose of certain diagnosis.

Complete loss of function due to anatomic interruption cannot be differentiated from loss of function due to physiologic interruption produced by compression, etc. By a single examination, it can be determined only whether the lesion is complete or incomplete. If complete, one cannot tell whether it is due to anatomic interruption or not, nor predict whether it will recover spontaneously or require surgical treatment. When subsequent examination reveals some return of function, and only then, can an incomplete lesion be diagnosed.

The author discusses the symptoms of complete interruption of a nerve as given by various authors and comments on the lack of standardized methods of examination; careless and verbose description and lack of knowledge of the physiology of the peripheral nervous system are exhibited by many authors on the subject. There are several degrees of loss of function, believed by the author to be the most important indications in determining the severity of a peripheral nerve lesion:

1. Complete loss of all functions of the nerve indicates a severe lesion and is interpreted as complete physiologic interruption of that nerve.

2. Total paralysis of all the muscles supplied by a nerve distal to a lesion cannot alone be used as an indication of the severity of that lesion.

3. Motion of segments about a joint does not indicate the integrity of the function of the nerve supplying the muscles ordinarily supposed to move such segments.

4. The preservation of certain movements, the loss of which is supposed to follow particular nerve lesions, may be supplemented, and lead to incorrect opinions that complete lesions are incomplete ones.

5. Only when sensation (pin prick) in the isolated supply of a peripheral nerve is present can the lesion be considered partial. In this area no overlapping of adjacent nerves occurs, and in complete lesions sensation to pin prick is absent. The author has been able to reconstruct the isolated supply of several peripheral nerves.

6. Complete reaction of degeneration is always present in severe lesions, but does not indicate an irreparable one.

The slowness of muscular contraction is the only constant phenomenon which can be satisfactorily employed in determining the reaction of degeneration. Polar changes are inconstant, and are frequently absent even in partial lesions. The rapidity and extent of muscle atrophy may be taken as a guide to the severity of a nerve lesion with certain reservations. The author compared the amount of atrophy by water displacement in partial and in complete lesions, the severe lesions showing a higher percentage loss of muscle substance but not enough to be of diagnostic value. Presence or absence of pain on pressure over the nerve distal to the lesion was found an unreliable test as was also pain on pressure over the paralyzed muscles. Loss of tone as measured by the tonometer is present only for a short time after the injury. Trophic disturbances are of value as an indication of the severity of the lesion when judged in the light of the presence of other conditions.

The signs of regeneration are different in spontaneous lesions and in those following resection and suture. In rapidly and spontaneously recovering lesions, pain and tactile sensation return simultaneously and in patches in isolated supply. There is no definite order in the return of the muscles, and it does not depend on the distance from the lesion. Slow and delayed cases that recover spontaneously act like sutured nerves, the muscles recover from above downward. The order in which signs of regeneration occur as given by Mme. A. Benisty are: (a) sensory regeneration, consisting of pain when the skin is pinched, pain when the nerve is pressed below the lesion, formication on pressure of the nerve and spontaneous aching in certain muscles; (*b*) arrest of atrophy and return of tonicity; (*c*) in some cases, return of faradic contractibility; (*d*) disappearance of objective sensory disturbance, and (*e*) voluntary movements.

In the author's experience, return of pain on pinching the skin can be used as an indication only when in the area of isolated nerve supply. A sensation of a different feeling often precedes other signs. Pain on pressure on the nerve trunk distal to the lesion was found unreliable. Tinel's signs were found to be practically valueless. Return of sensibility to pain has always been accompanied by return of sensibility to touch in the isolated supply of a nerve. The author believes that return of sensibility to pain without sensibility to touch only occurs in the area of nerve overlap and is due to the overlap in the area of anatomic sensory supply.

In primary suture, the first return of motion in musculospiral cases was six months; in external popliteal, seven months; in sciatic, six and one-half months. In secondary suture, motion returned in the musculospiral in from five to six months, and in the sciatic in six and one-half months. The author regards as a certain sign of nerve regeneration the disappearance of the reaction of degeneration, the return of objective sensibility in the isolated supply of a peripheral nerve, and the return of motion.

Ott, Rochester, Minn.

LA PSYCHO-ENCEPHALITE AIGUE EPIDEMIC ET LES TROUBLES PSYCHIQUES DE L'ENCEPHALITE AIGUE, DITE LETHARGIQUE (EPIDEMIC ACUTE PSYCHO-ENCEPHALITIS AND THE PSYCHIC DISORDERS OF ACUTE, OR SO-CALLED, LETHARGIC ENCEPHALITIS). Hesnard, Encéphale **15**:443 (July 10) 1920.

Concerning the mental symptoms in epidemic encephalitis, the writer distinguishes two clinical groups: first, the mental symptoms that arise in encephalitis in its common (and numerous) forms (oculolethargic, polymyelitic, neuritic, tetanic, choreic, myoclonic, etc.), and second, the mental symptoms in those so-called encephalitic psychopathies which Hesnard chose to name epidemic acute psycho-encephalitis.

Mental symptoms if not constant are at least frequent in epidemic encephalitis. The "sleep" in these cases resembles a veritable "twilight state," analogous to certain hypoconfusional syndromes. It is more a "psychosis of closed eyes" than true sleep.

The large group which Hesnard thinks of as encephalitic psychopathies, and would keep quite separate, is divided into five subgroups in his experience: 1, psychosomnolent; 2, lucid catatonic stupor; 3, acute delirious types; 4, confusional forms properly speaking, and 5, Korsakoff types.

1. Psychosomnolent forms. These cases, on a basis of hypersomnia, accompanied by a neurologic syndrome, mesencephalic or otherwise, develop psychic disturbances, often consisting of slight excitement, or sometimes hypomania, passing into confusion. Then the patient passes into a clearly depressive stage. In some instances, the confusion, and with it a more or less massive intel-

lectual obnubilation, arises so brusquely as to give clinically the picture of general paralysis during the invasion period.

2. Forms with lucid catatonic stupor. The patient is impassive, the expression vacant. There is simple catatonia. He has lost "psycho-minic mobility." The life of his physiognomy, as his general muscular activity, has been suspended. There is a viscosity of voluntary activity. The catatonia is genuinely psychomotor and not (as occurs in other pseudoparkinsonian cases) primarily explained by a muscular rigidity alone. In other words, its psychic factor is actual. But, and the writer says it is a curious thing, these patients are nevertheless habitually lucid and cannot be said to be confused. In total, it may be said regarding catatonia in encephalitis that one kind seen can be regarded as true, and does not lack a psychic basis. The other kind is false inasmuch as it is merely a more or less necessary result of muscular rigidity.

3. Acute delirious forms. The delirium may be frankly hallucinatory, with sometimes an alarming general state. Even when collapse threatens, it is rare to find meningeal reactions. In brief, this state is analogous to a febrile delirium tremens.

4. Confusional forms, properly speaking. "The mental confusion which is most frequent is a psychic torpor, which, when it becomes ameliorated to a degree, shows itself in the form of a bradypsychic syndrome." (In this section, in spite of its title, the author fails to talk about confusion. The latter is really discussed in Subgroup 5, which follows.)

5. Korsakoff forms. The Korsakoff syndrome (with disorientation and a certain degree of confabulation) is encountered in prolonged forms of diffuse encephalitis, sometimes accompanied by neuritic symptoms or more frequently still by poliomyelitic symptoms. The symptoms usually represent either a bipolar psychoneuritic association or a bipolar psychomedullary association. There are instances without either neuritis or myelitis. From this, one concludes that the Korsakoff syndrome is not obligatorily neuritic. Certain psychopathic sequels belong here, comprising a state of psychic enfeeblement. Without interest, curiosity or initiative, though conversing voluntarily and with good sense, one patient continually believed herself to be in another city. She took unto herself no new memories and made no effort to "grow precise regarding her situation." It was a condition comparable to those cases (with a destructive lesion, arterial or otherwise) in which the connections of the frontal lobes with other cortical areas are interrupted.

Psychic sequels sometimes assume other forms, especially in children. In them Cruchet of Bordeaux has found intellectual inappetence, difficulties of learning, and also disturbances of character and disposition, the permanency of which only the observations of the future will establish.

Davis, New York.

THE ROLE OF DEEP ALCOHOL INJECTIONS IN THE TREATMENT OF TRIGEMINAL NEURALGIA. Harvey Cushing, J. A. M. A. **75:** 441 (Aug. 14) 1920.

The author reviews thoroughly the history of deep alcohol injections in the treatment of trifacial neuralgia. Injection of the nerves with some tissue fixative was first practiced as a means of controlling neuralgias. Various substances were used for this purpose. Chloroform was introduced by Bartholow in 1874, and osmic acid was proposed ten years later by Billroth and Neuber. Pitres and Verger of France were the first to introduce treatment by the injection of alcohol; further developments in this method were made by Levy and Badoin, and later by Patrick.

The results obtained from alcohol injections are necessarily limited. The relief is only temporary, and, with each successive injection, a shorter interval of freedom from pain is likely to be secured. The procedure is, however, less hazardous than avulsion of the peripheral branches. Paralysis of the oculomotor nerves is the most common complication. Stiffness of the jaw, paralysis of the motor fifth, sloughs into the soft tissues and bones, hemorrhage, and facial paralysis are other possible complications; accidental injection into the middle ear is not unheard of, and the resulting trouble is most distressing.

The author condemns injection of the ganglion, since it is impossible to control the extent of alcohol infiltration, and the postoperative complications are most serious. Motor paralysis of the fifth, secondary keratitis, and meningitis are the most frequent complications.

His conclusions are:

1. Deep extracranial injections of alcohol into the maxillary and mandibular nerve trunks near their foramina of exit from the skull have completely superseded peripheral neurectomies.

2. In neuralgias which are limited to one of the two lower divisions and which may possibly not extend into the other trigeminal areas, alcohol injections represent unquestionably the treatment of choice.

3. When the neuralgia has spread beyond its original area and come to involve that supplied by the adjacent division, a trigeminal neurectomy must be contemplated; but if no preceding deep injection has been given, it may be useful not only in insuring the type of the neuralgia but in giving the patient some warning as to what the numbness resulting from the neurectomy may amount to.

4. Deep alcohol injections are sometimes useful, furthermore, in determining in doubtful cases whether the syndrome is a true neuralgia of the tic douloureux type or one of the peculiar and rare pseudoneuralgias not amenable to relief either by injections or neurectomies.

5. Even the extracranial injections are not entirely free from risk, and in no case should they be pushed to the point of attempting an injection of the gasserian sheath itself.

6. In consideration of the perfect and permanent results secured today by a trigeminal sensory root avulsion, the prolonged and repeated use of injections in refractory cases which involve more than one division should be deplored.

Adson, Rochester, Minn.

LA CONFUSION MENTALE D'ORIGINE PUERPERALE (MENTAL CONFUSION OF PUERPERAL ORIGIN). R. Dupouy, Bull. méd. **34**:722 (Aug. 14) 1920.

Puerperal psychoses are the psychoses due to auto-intoxication or infection coincident with pregnancy or the puerperium, having confusion, in one of several varieties, as the type clinical form. Certain psychopathic manifestations constitute an apparent exception to this rule. A number of women during their pregnancy develop "envies" (inclinations) sometimes even impulsions. These are puerile or absurd desires, the nonsatisfaction of which provoke phenomena of anxiety. Related to these are such fears as of bringing into the world a child which is "marked." Such manifestations are not accompanied by confusion. They are on a par with the headaches and vomiting of pregnancy, and appear along with the classic symptoms of gravid autointoxication. Such "envies" correspond in rough sketch to the mental automatism which may succeed them, just as the accompanying somnolence, headache, lassitude, etc., represent in preliminary outline the confusional syndrome itself.

The two main groups of the confusional syndrome are the true manifestation of puerperal psychosis—mental confusion by autointoxication—and mental confusion dependent on puerperal infection. The mental confusion by autointoxication appears in the third or fourth month of pregnancy and by the seventh month assumes the intensity which forces special care. At first, there is a simple embarrassment of voluntary exercise of the intellectual faculties, and somewhat later, more and more memory errors. Gradually, the patient fails to understand well what is said to her. She speaks hesitatingly, asks childish questions and has vague ideas. Some arrive at mutism, confusional stupidity and catatonia. In other cases various ideas of persecution and of influence make their appearance. Delirium is fed by hallucinations, chiefly visual, and often a paroxysm of hallucinations lets loose a reaction, anxious or impulsive.

In the gravid woman an apathetic mental confusion is the rule. It is after the delivery that the restless hallucinatory delirium installs itself. If the latter arises, it is always possible to trace back and find the former during the actual gestation.

The author advises thyroid opotherapy for cases that occur during the gestation period but in the cases following delivery he advises ovarian therapy which has been preceded by thyroid treatment.

As regards the second group, the writer believes that confusional states are induced by puerperal infection only in women who are constitutionally predisposed. In these cases confusion is accompanied by wild agitation. The patients are delusional and hallucinatory and are in danger of harming themselves. He has never seen a Korsakoff type of psychosis occasioned by an infection in the puerperium.

The diagnosis of a puerperal confusional state is easy when the signs of infection are evident and the history is open. When there has been a clandestine abortion, the situation is more difficult. This emphasizes the need of a gynecologic examination, and indicates the needed surgical care of the patient in conjunction with psychiatric oversight.

DAVIS, New York.

UEBER MYOTONISCHE DYSTROPHIE (MYOTONIC DYSTROPHY).

KARL HITZENBERGER, Monatschr. f. Psychiat. u. Neurol. **47**:249 (May) 1920.

Under this heading the author reports a case, with postmortem findings, which belongs to the group of atrophic myotonia (Steinert). He prefers the term myotonic dystrophy, from the belief that these cases are primary dystrophies, with a secondary, symptomatic myotonia. According to Steinert's description, such cases show: myopathic facies, atrophy of muscles of forearm and hand, of sternocleidomastoids, of lower extremities and of the bulbar muscles, myotonic reaction, partial baldness, atrophy of testicles and vasomotor disturbances.

Hitzenberger's patient was 38 years of age. The family history was negative. Onset occurred at the age of 24 with myotonic symptoms in the small hand muscles. The fully developed picture showed: myopathic facies; extensive dystrophic changes in the muscles of the forearms and legs, with absence of such changes in the upper arms and thighs; myotonic changes only in the thenar muscles, which in addition to the myotonic reaction, also showed a mysthenic reaction. Death resulted from pulmonary tuberculosis.

The necropsy findings were: Brain: no gross or microscopic changes. Spinal cord: number of cells considerably reduced; many cells small, shrunken, and poor in tigroid bodies. Some cells showed pigment increase. The author found no evidence of degeneration of the fiber tracts, in distinction to Steinert, who found tabiform degeneration in the posterior columns and no cell changes. Muscles: an elaborate description is given of the histologic changes. The affected muscles showed hypertrophy, atrophy and degenerative changes. There was an enormous increase in the number of muscle nuclei, which lie not only in the periphery but scattered through the whole body of the fiber. There was extensive increase in the fibrous connective tissue. Atrophic changes in testicles: no changes in parathyroids, thyroids, suprarenals, pancreas or thymus.

Selling, Portland, Ore.

SUR LES NEVRALGIES DE LA TETE ET LEUR TRAITEMENT PAR L'ECRASEMENT DES POINTS DOULOUREUX (NEURALGIAS OF THE HEAD AND THEIR TREATMENT BY COMPRESSION OF THE PAINFUL POINTS). W. Janowski, Presse méd. **28:**537 (Aug. 7) 1920.

Trigeminal and occipital neuralgias often appear in the guise of "ordinary," that is, "vasomotor" headaches, and their true nature is not detected because of neglect to palpate the points of emergence of nerves which should be under suspicion. If pressure is applied over each of these points during or immediately after the headache, a resulting violent and radiating pain throughout the area of distribution will determine the presence of neuralgia in the branch tested. Headaches reacting positively to this test, even some migraines, together with frank idiopathic neuralgias, can be grouped as available for the compression method of treatment. One is warned against including any neuralgias secondary to a demonstrable pathology — dental, intracranial, etc.

The therapeutic technic is as follows: The operator holds the patient's head firmly with one arm, keeps a finger of his other on the painful point, and exerts very strong pressure on it for one-half to one second, repeating the compression ten times at intervals of one second. This treatment is given once a day for a series of six successive days; the series is repeated several times with two resting days intervening. From five to ten series are found to effect a cure in obstinate cases of long standing. Early cases have recovered in ten or twelve days, with less than two series of treatments.

The severe pain associated with such a procedure is admittedly a handicap, but is partly overcome by the concomitant administration of acetylsalicylic acid, codein or other sedatives for the first few days. An alcohol injection is much more painful, and need not be given except as a last resort, if the compression method is accorded a fair trial.

Huddleson, New York.

THE LATE TREATMENT OF GUNSHOT WOUNDS OF THE HEAD.

H. H. Kerr, Surg., Gynec. & Obst. **30**:550 (June) 1920.

The author emphasizes the advantage of using osteoperiosteal grafts in relief of the "defect syndrome" presented in cases of skull wounds which involve the brain. While very little is accomplished in the presence of organic brain lesions, a group of symptoms, constituting the defect syndrome, is relieved by removal of the bone deformity.

The author says of patients suffering from skull defects: "They are morose, retiring, and avoid their fellows. They suffer more or less from headache. Stooping over or turning suddenly will produce dizziness. Loud noises are extremely irritating. Exposure to the sun produces headache and prostration. Perhaps the fear of injury and the knowledge of incomplete protection to their brains contribute to this characteristic train of symptoms."

The author advocates the use of local anesthesia. The requirements of successful bone grafting are best obtained by sewing the graft to the pericranium with the bone surface inward. Operation should be delayed for at least three months after healing; in cases of osteomyelitis of the skull, it is advisable to postpone bone grafting until at least six months after healing of the wound. Regulated and directed exercise together with massage of the partially paralyzed extremities should be instituted, preferably in connection with play, since better cooperation is obtained in this way than if prescribed as a strictly therapeutic measure.

Plastic surgery on skull defects is at best unsatisfactory. Although some patients gradually improve, the condition of many remains unchanged. Unless the patients have a very large and disfiguring defect, operative interference should not be enthusiatically urged.

Adson, Rochester, Minn.

NORMS FOR SOME STRUCTURAL CHANGES IN THE HUMAN CEREBELLUM FROM BIRTH TO OLD AGE.

Robert S. Ellis, J. Comp. Neurol. **32**:1 (Aug. 15) 1920.

Neuropathology has always suffered from the handicap of inadequate knowledge of the healthy norms. The comprehensive studies of growth at the Wistar Institute are contributing valuable data in this field. Ellis has undertaken a study of the human cerebellum from this standpoint. In a paper published in the *Journal of Comparative Neurology* (vol. 30, 1919) he reported the results of quantitative study of the Purkinje cells in normal, subnormal and senescent persons, finding that the number of these cells is deficient in idiots and imbeciles and that the number decreases progressively in old age. These data are considerably amplified in Ellis' recent article, with tables and graphs showing the following relations:

At birth the cerebellum is small in comparison with the cerebrum, but at the age of 15 months the parts of the brain have approximately the same relative weights as those of the adult. The relative weight of the cerebellum does not vary significantly with stature, sex, race or intelligence. Between birth and the age of 15 months the layer of external granule cells disappears, the Purkinje cells grow to normal adult size, the molecular layer and internal granular layer assume the same relative widths as found in the adult, and the myelin sheaths grow rapidly, especially at the end of the period. These changes are to be correlated with the fact that about this time the motor activity of the child is increasing rapidly and he is beginning to walk. With the beginning of senescence there is a loss of Purkinje cells and a resultant loss of myelinated fibers. With the structural change is to be correlated the loss of strength and of muscular coordination which is characteristic of old age. There is some indication that in senescence the right cerebellar hemisphere loses more cells than the left, due perhaps to the effects of excessive use of the right half of the body.

Herrick, Chicago.

PRESENTATION D'UN OCULO COMPRESSEUR MANOMETRIQUE (A MANOMETRIC OCULO-COMPRESSOR).. J. Roubinovitch, Progrès méd. **35**:367 (Aug. 21) 1920.

The author has perfected the ocular compressor which he originated in 1916, and he describes it in detail. A leather band fitted about the head holds in place over each eyeball an oval elastic air chamber. These are inflated by means of an ordinary hand bulb placed at the terminal of an attached T-shaped set of rubber tubing. A spring-manometer, reading in centimeters of mercury, is inserted in the system. A three-way stopcock at the point where this is inserted permits one to keep constant pressure on the eyeballs for a time if that is desired. Other stopcocks make it possible to compress one eye at a time.

When the apparatus is in place over the eyes, using the manometer as a constant guide, one raises the pressure to a point not above 30 or 35 cm. of mercury. With a certain degree of ocular compression, variable with each individual, one obtains oscillations of the needle on the manometer synchronous with the pulse.

The real function of the apparatus lies in the study of the oculocardiac reflex, and by it curves can be charted showing the cardiac pulsations per minute under the influence of a series of compressions of different intensities.

Mougeot, in 1914, and others since, have shown the therapeutic value of the oculocardiac reflex in tachycardias, in (certain) tremors and in abnormal movements (athetoid).

Davis, New York.

UEBER ZOANTHROPY (ZOANTHROPY). C. Schneider, Monatschr. f. Psychiat. u. Neurol. **48**:269 (May) 1920.

The patient, a woman of inferior mentality, aged 31, developed an anxiety state following roentgen-ray treatment (for uterine hemorrhage). Hysterical attacks and a hysterical dream state followed. During the latter she saw three dogs which were trying to devour her. She developed an outspoken zoanthropic attack—imitating the actions and barking of a dog.

The author reviews briefly the literature on the subject, both ancient and modern, referring especially to the outbursts in epidemic form. He separates the cases into two groups:

1. Simple imitation of animal sounds, due to a respiratory muscle cramp, analogous to other types of hysterical spasm; no clouding of consciousness.

2. Zoanthropy; imitation of animal sounds and acts, accompanied by clouding of consciousness. These are closely related to the hysterical dream states. A fear element is practically always involved. Between the psychic trauma and its conversion into a zoanthropic attack, a latent period elapses. At first the outbursts are brought on only by something that can arouse a memory

picture of the original exciting incident, but later the associations leading to attacks become more remote, and finally the attacks become independent of external factors. The author attempts to make an analysis of the origin, development and fixation of the attacks, with special reference to: (1) the relationship to occurrences of normal life, and (2) the special reasons for its development in hysteria.

Selling, Portland, Ore.

THE EXTENT OF THE FLOOR PLATE OF HIS AND ITS SIGNIFICANCE. B. F. Kingsbury, J. Comp. Neurol. **32**:113 (Aug. 15) 1920.

This paper proposes an important modification of the plan of morphologic analysis of the brain suggested by His. A rational basis for the division of the brain into epichordal and prechordal portions is afforded. The floor plate of the neural tube is primarily coextensive with the notochordal plate and the floor plate corresponds with a sutura neurochordalis (His). The neurochordal suture does not extend to the anterior end of the neural plate and the primary sensory and motor zones are continuous from side to side cephalad of the anterior end of the floor plate. This involves a reinterpretation of the morphology of the forebrain and midbrain. The floor plate is regarded as terminating in the fovea isthmi at the anterior end of the midbrain floor; the motor zones terminate with the midbrain; the boundary between sensory and motor zones terminates near the mammillary recess; the optic chiasma marks the anterior end of the neural plate; the sensory zones extend farther forward than do the motor zones; and the region from which the retinas are derived lies in the most rostral part of the neural plate. The last conclusion is correlated with the studies on experimental cyclopea of Stockard.

Herrick, Chicago.

MENTAL DEFICIENCY; ITS FREQUENCY AND CHARACTERISTICS IN THE UNITED STATES AS DETERMINED BY THE EXAMINATION OF RECRUITS. Pierce Bailey and Roy Haber, Ment. Hyg. **4**:564 (July) 1920.

Neuropsychiatric examiners of the Medical Corps identified 72,323 cases of nervous and mental disorders, and of this number 22,741, or 31.4 per cent., were mental defectives. Only those who are familiar with mental deficiency in all its phases will be able to estimate the tremendous economic asset and gain in morale to the A. E. F. which is represented by these statistics. It is interesting to note that in nineteen states the percentage of defectives was above the average for the entire United States, and that all but four of these states were southern. The negroes were listed separately, and in seven states the average was exceeded. Maryland showed 74.4 per cent. Of the whole group 71.8 per cent. were native whites and 18.7 per cent. native negroes, and the remaining 9.5 per cent. were foreign born. Of this last subdivision, 32.5 per cent. were Italians and 32.1 per cent. Russians. Great Britain and Switzerland were the only two countries that had under 10 per cent. of mental defectives.

The neuropsychiatrist, the psychologist and the sociologist will all be repaid by a careful study of this authorized report from the Surgeon-General's Office.

Strecker, Philadelphia.

SINUSITE FRONTAL AIGUE ET PTOSIS (ACUTE FRONTAL SINUSITIS AND PTOSIS). G. Liebault, Bull. méd. **34**:815 (Sept. 11) 1920.

In three instances recently the writer has seen acute unilateral frontal sinusitis associated with ptosis. Usually ptosis which develops during a sinusitis is due to the existence of infection of the perisinus tissue, with edema of the lid and other conditions. The writer has deemed his cases worthy of special report as in them the ptosis had no such basis; rather the lid was entirely free from edema. Its loss of normal movement was neural and was occasioned by a certain temporary involvement of the nerve fibers which pass along the floor of the sinus and enervate the levator. The author shows that there was no cellular infiltration, no conjunctival irritation or chemosis, no exophthalmos and no disturbance of the movements of the eyeball. Unfortunately, in two of the cases the fundi were not examined. In one case he reports an obscurity of the disk on the affected side. The infection of the sinus was purulent and outspoken.

The ptosis endured about ten days and disappeared as the sinusitis was relieved. This, in turn, was accomplished by medical measures, and surgical procedures were not followed in any instance.

Davis, New York.

COMPARATIVE STUDIES ON THE GROWTH OF THE CORPUS CALLOSUM. Nobuharu Suitsu, J. Comp. Neurol. **32**:35 (Aug. 15) 1920.

In this first paper of a series the area of the corpus callosum as measured in sagittal section was studied in the albino rat at successive ages. Myelinated fibers are not present at birth; they appear at the seventh to tenth day. The area of the section increases between birth and maturity about three and a half times. This increase occurs in three phases which correlate with the functional development of the rat. During the first ten days of life new unmyelinated fibers are added. From the tenth to the thirtieth day the formation of new myelin sheaths is the main factor. After the thirtieth day the fibers increase in diameter, the increase continuing as long as the brain increases in weight.

Herrick, Chicago.

EXPERIENCES WITH LUMINAL IN EPILEPSY. Julius Grinker, J. A. M. A. **75**:508 (Aug. 28) 1920.

Grinker comments favorably on luminal as a convulsion controlling agent in essential epilepsy. In a series of 100 cases the seizures practically ceased for periods of time varying from several months to four years. The average dose was 1.5 grains, but if necessary double and even triple this amount was given. There seem to be no untoward effects beyond the occasional appearance of a slight rash. Apparently the drug does not produce mental sluggishness nor is it habit forming. In general, the author's results bear out the experience of Dercum and other observers.

The reviewer takes this opportunity of citing his own experience. Luminal has been extensively employed in the clinic for mental and nervous diseases of the Pennsylvania Hospital. It is effective not only in idiopathic epilepsy, but also in cases in which syphilis or other factors may be demonstrated. However, the distinction between so-called "essential" and other forms of epilepsy must often be largely theoretical and the exhibition of luminal should not negative the effort to search for and correct possible causative factors. There is some reason to believe that this drug may have a direct influence on motor centers. In three cases of chorea the movements were promptly inhibited in two by luminal medication.

STRECKER, Philadelphia.

THE HEMORRHAGIC TENDENCY AS A FREQUENT CAUSE OF CRANIAL HEMORRHAGE OF THE NEW-BORN. JOHN A. FOOTE, Am. J. Dis. Child. **20**:1 (July) 1920.

In this article the author has followed the line of reasoning which he expressed in June, 1918, that many of the cases which do not show rupture of the meningeal artery are not frank hemorrhages so much as oozing of the vessels of the pia, and that great relief can be obtained by lowering intracranial pressure through lumbar puncture with some method to increase the coagulability of the blood, such as blood serum, citrated blood, etc. Within the last fifteen months, this procedure was followed at the Providence Hospital in Washington, D. C., in seven cases. In all new-born infants, showing, within from twelve to twenty-four hours after birth, respiratory distress and blueness with or without muscular rigidity and twitching, lumbar puncture was performed, and in every instance with these symptoms the lumbar puncture showed blood varying from a slight tinging to clots. Horse serum and thromboplastin were used hypodermically. One infant died; the other six have been traced and are still living. To corroborate his findings, the author quotes the clinical observations of Dr. Margaret Warwick who in thirty-six necropsies on still-born infants or on those who had died within a short period after birth found that 50 per cent. had hemorrhages in the dura, brain surface or ventricles; only one was a case in which forceps had been used. Two out of eighteen were born dead. In eight, or 50 per cent., of those showing cranial hemorrhage, hemorrhage was found in other organs. He quotes a case of a baby delivered with forceps after a long labor who two hours after birth became blue, and whose respirations were labored; coagulation time exceeded nine minutes; lumbar puncture revealed small clots. The child also vomited red blood, and blood came from the rectum, nose, stomach and ear. It was given horse serum and thromboplastin and recovered. The author finds, following Cushing and Sharpe, that simple surgical relief of pressure when a focal diagnosis can be made yields brilliant results and avoids the horrible paralysis known as Little's disease. An attempt to increase the coagulation time should be made prior to operation.

The author does not lose sight of the fact that hemorrhage may occur in the infant's skull, and without treatment may be followed by complete recovery. He makes a plea that routine coagulation time tests be made on the new-born infant.

WINKELMAN, Philadelphia.

EFFICIENT TREATMENT OF EPILEPSY BY LUMINAL. GASTON MAILLARD, Encéphale **25**:455, 1920.

Maillard, following the publication of Raffegean (*Société médico-psychologique,* Dec. 29, 1919), used luminal in all the epileptics on his service. He recalls that barbital (veronal, diethylmalonylurea) only differs from luminal (phenylethylmalonylurea) by a phenyl group. Since barbital has no effect on epilepsy and luminal has, it would seem that the phenyl group is the determining factor.

Pecheux first noted the good effect of luminal in epilepsy in 1913, but it was not tried so much as an anti-epileptic as a depressant for certain excited phases of the disease. Frankenhauser noted good results in 1915. Maillard cautions that at first the dosage must be carefully watched. He starts with morning and evening doses of 10, 15 or 20 cg. (1/6, 1/4 or 1/3 grain). Later the dose may be diminished to 5 cg. The most prominent symptom at the beginning is sleepiness. The patient awakens with difficulty in the morning and tends to fall asleep during the day. In a few cases there is a period of euphoria. These initial symptoms soon disappear. If, for some reason, the treatment is to be stopped, it must be done slowly, else there may be a sudden increase of severe attacks. Patients with psychic symptoms may show an exaggeration of these when the convulsion begins, diminishes or ceases, which Maillard interprets as a transference of the epileptiform explosion. Chronic psychopathic conditions, on the other hand, are improved by luminal.

Maillard used the drug for months without any ill effects, thus stopping the seizures and bettering the general psychic condition. He does not say whether the cure is permanent but promises another communication on this point after a longer experience with the drug.

In the discussion, Dr. Laignel-Lavastine mentioned a patient under luminal treatment for four years, in whom the motor seizures had ceased but who had severe spells of anger. Dr. Hartenberg mentioned two cases treated by luminal for from six to seven years with similar attacks.

Kraus, New York.

THE CONDITIONS IN THE BRAIN DURING STAMMERING. Editorial, J. A. M. A. **75**:608 (Aug. 28) 1920.

The theory of Bluemel that "stammering is caused by transient auditory amnesia in the auditory or speech center brought on by cerebral congestions" has been definitely established as a fact by the experimental work of Robbins.[1] The subject was a person who had sustained a bullet wound of the right frontal lobe without injury to the cerebral tissue. By means of a glass plethysmograph applied at the site of a small trephine opening, the increase in brain volume accompanied by an increase in the size of the pulse which occurred under conditions of pronounced shock, fear of stammering and other emotions, were noted and recorded. During intervals of normal reading the brain of the stammerer receded to its normal volume. "Evidently the fear of stammering and the forcing out of hard words which accompanies stammering causes vasoconstriction in the periphery and cerebral congestion. As Robbins expresses the situation, the congestion blurs verbal imagery, especially auditory verbal imagery, and makes it impossible for the stammerer for the time being to recall a part or the whole of the word he wishes to speak at the moment he has to say it." It is evident that in order to cure stammering both the fear and the accompanying abnormal muscular contractions must be overcome.

Strecker, Philadelphia.

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[1] Robbins, S. D.: A Plethysmographic Study of Shock and Stammering, Am.J.Physiol. **48**:285 (April) 1919.

ON THE ORIGIN OF THE GANGLION CELLS OF THE NERVUS TERMINALIS OF THE ALBINO RAT. F. W. STEWART, J. Comp. Neurol. **32**:99 (Aug. 15) 1920.

The recent demonstration of the presence of a distinct terminal nerve in close association with the olfactory nerve in all vertebrates from fishes to man continues to stimulate investigation regarding its connections and probable functions. In view of recent evidence tending to relate this nerve with the sympathetic system, the author has studied the development of its ganglion cells, confirming the conclusions of Brookover concerning fishes and the less definite results of earlier students in mammals that the ganglion cells of this nerve are derived by proliferation inward from the embryonic olfactory epithelium. None of these ganglion cells seem to arise from any of the sources usually recognized as giving rise to sympathetic neurons.

HERRICK, Chicago.

THE SIGN OF THE SUPRACLAVICULAR FOSSA (PARALYSIS OF THE SERRATUS MAGNUS FOLLOWING INFLUENZA). GEORGE GUILLAIN and E. VIBERT, Ann. de méd. **7**:383, 1920.

These authors present a case of isolated paralysis of the serratus magnus muscle following influenza, in which, besides the familiar signs of this condition, there existed a definite difference between the supraclavicular fossa on the well and affected sides. The sign appears on elevating or shrugging the shoulders. The fossa on the affected side was much narrower, measuring laterally from the midline. The clavicle appeared to be pulled upward and toward the middle of the back. The depth of the fossa on the affected side was diminished, due to filling up by the sternocleidomastoid muscle. Instead of a triangular space bounded by this muscle, the trapezius and the clavicle, only a narrow gutter-like space existed. This sign is of diagnostic interest and adds to our knowledge of a comparatively rare condition.

KRAUS, New York.

THE MOTOR NUCLEI OF THE CEREBRAL NERVES IN PHYLOGENY—A STUDY OF THE PHENOMENA OF NEUROBIOTAXIS. III. REPTILIA. DAVIDSON BLACK, J. Comp. Neurol. **32**:61 (Aug. 15) 1920.

This is one of a series of studies on the comparative anatomy of the motor nuclei of the cranial nerves from the point of view of the functional factors correlated with phylogenetic changes in their arrangement. In reptiles, as compared with still lower forms, the factors chiefly significant in this connection are the perfection of the apparatus for air breathing, the development of a mobile fleshy tongue, increased complexity of the jaw muscles and greater refinement of the sense of sight. There is considerable variation in the arrangement of these nuclei among the different groups of reptiles, and these differences are also correlated with diversity in modes of life.

HERRICK, Chicago.

ENDOCRINE IMBALANCE IN THE FEEBLEMINDED. Oscar Raeder, J. A. M. A. **75**:527 (Aug. 21) 1920.

The author has attacked this problem from the angle that is least open to criticism. He studied 100 cases of feeblemindedness from the standpoint of the necropsy findings. The results throw a new light on endocrine pathology in mental deficiency. In ten instances, "extreme," in eleven, "marked" and in fifty-three "moderate" glandular changes, including aplasia, hyperplasia, pigmentation and interstitial changes, glandular proliferation and various quantitative anomalies, were revealed both by gross examination and under the microscope. The pituitary gland was involved most frequently and was closely followed by the sex glands. The only weak point of Raeder's contribution is the absence of serologic investigation in many of the cases and the consequent failure to exclude completely a possible syphilitic factor.

The value of this type of work cannot be over-emphasized. It should not only stimulate similar investigations, but should also furnish an incentive for intensive clinical and experimental therapeutic research.

Strecker, Philadelphia.

SUR L'ENCEPHALITE LETHARGIQUE (LETHARGIC ENCEPHALITIS). Léon Bernard and Jules Renault, Informateur d. alién. **15**: 174 (June) 1920.

Bernard and Renault presented to the Paris Academy of Medicine, May 18, the results of a national survey of lethargic encephalitis. In the fifty-five departments reporting, there had been 70 cases in January, 206 in February, 144 in March and 44 in April—465 in all. Children seem to have been affected half as often as adults, and persons over 60 were affected very rarely. The mortality had been 10 per cent. Cases had invariably been rather widely scattered, no direct contagion being observed. The disease has generally been thought to be spread by carriers. All reports were unfavorable to the idea of a relationship between poliomyelitis and lethargic encephalitis, but the probability of some connection between encephalitis and grip has often been noted.

Huddleson, New York.

INFLUENCE OF WAR ON CONCEPTS OF MENTAL DISEASES AND NEUROSES. Sidney J. Schwab, Ment. Hyg. **4**:654 (July) 1920.

Schwab attempts to evaluate the material gains to neuropsychiatry which have resulted from the wholesale war experiment of bringing the complex and highly developed human nervous system into contact with destructive forces and conditions. He points out that we have every reason to expect additions to our knowledge of the physiology of the peripheral nerves and the central nervous system. In the field of psychoneuroses the net gain should be a better understanding of mechanisms and a consequent stimulation of logical methods of therapy. The relative curability of episodic "war psychoses" should encourage the psychiatrist in dealing with the psychoses of civil life. The final, and perhaps most important, fact is that awakened and stimulated professional and public interest practically guarantees an earnest effort to apply our war won knowledge to all neuropsychiatric problems of civil life.

Strecker, Philadelphia.

Society Transactions

NEW YORK NEUROLOGICAL SOCIETY

The Three Hundred and Eighty-Second Regular Meeting, Oct. 5, 1920

WALTER TIMME, M.D.. *President*

A CASE OF LETHARGIC ENCEPHALITIS. Presented by DR. ISADORE ABRAHAMSON.

An unusual case of lethargic encephalitis was presented for the purpose of showing how intensive study of this disease had shed light on many obscure points in the pathology of diseases of the nervous system. New names are frequently attached to unusual variations of one disease, and discoveries of distinct disease entities are heralded without cause. The patient illustrating the complexity of encephalitis manifestations was an Italian butcher. His previous personal history was negative. The initial symptoms were pain in the mastoid, left occiput and left shoulder. There was fever for three days, followed by a lethargic sleep for two weeks, from which he was easily aroused, answering in a typical fashion. Movements set in, being first present in the left shoulder and afterward in the left index finger. The other hand was soon affected and the speech became markedly involved, being dysarthric, not aphasic. There was no fine tremor. The legs were involved later. Pains and paresthesia in the suprascapular region and nape of the neck have persisted from the time of the onset nine months ago until the present. The patient complained of formication about the left shoulder. Neurologic examination showed the right pupil ovoid up and down and sluggish to light; accommodation was normal. The left pupil was ovoid from side to side; accommodation was not so good; it was sluggish to light.

The most marked feature exhibited by this patient was the nature of the movements of the upper extremities, the left especially. The movements were slower than those of chorea and resembled intentional movements, being slow, coordinated and asymmetrical. There was no fibrillation or myoclonia. Every movement of a muscle is followed by movement of a part, both antagonistic and synergic. There is thus presented in this patient, as a late sign of an undoubted lethargic encephalitis, a disturbance of motility, without ataxia, without loss of power, atrophy or dysmetria, showing no dyssynergia or myoclonia. There are evidences of sensory irritation still persisting. The movements are neither choreic nor paramyoclonic, nor do they resemble those of dyssynergia cerebellaris. They are not hysterical. They are not the movements of dystonia. There is no torsion spasm of the pelvis or neck. They are extrapyramidal, not induced by pain or paresthesia. It is impossible to designate the affliction by any accurate form in the recognized categories. The accepted classification of disease according to manifestations is here clearly seen to be untenable.

A CASE OF LETHARGIC ENCEPHALITIS. Presented by Dr. Thomas K. Davis.

The patient was a young woman in whom encephalitis had been a complication of pregnancy. The encephalitis began three weeks before the birth of a child in February, 1920, and quite early took the pseudoparkinsonian form. With this, the arms assumed a constant tonic contracture across the chest, each elbow being strongly flexed. They could be passively extended only with great difficulty. The physician who saw her at that time placed the arms in full extension in plaster casts, which were worn for three weeks. This resulted in the arms becoming rigid at full extension. It has been quite impossible to flex either elbow since.

There is usually a fine tremor of the hands. There have been no pyramidal tract signs.

A roentgenogram of the right arm showed a faint suggestion of new bone formation at the elbow, possibly beginning ankylosis. In the left arm, bony changes were absent and the high degree of muscular tone apparently alone produced the rigidity of this arm.

DISCUSSION

Dr. Foster Kennedy pointed out that this second case was practically the reverse of that shown by Dr. Abrahamson. There was apparently overtoning of the cervical muscles. The trunk muscles were not affected.

Dr. Isadore Abrahamson called to mind a patient with spondylos rhizomelique who exhibited the same pains, attitude and rigidities as the second patient, in whom the condition had been the result of immobilization. There were, however, no signs of cord or brain implication. Dr. Abrahamson regarded the case presented as both lethargic and spondylotic.

Dr. C. B. Craig had seen the first patient at the Neurological Institute for a period of two weeks in June. His condition had not changed from that time. It was observed that the motility ceased during sleep. Faradism had proved to be beneficial, its effects lasting from one to three hours. This was probably a fatigue phenomenon. The case was diagnosed as chorea following lethargic encephalitis.

Dr. Bernard Sachs remarked on the interest attached to two such diametrically opposed cases. He said he hoped to gain anatomic information from them and was especially interested to know what part of the brain was responsible for the disturbances of motility. The condition of the last patient resembled certain infantile hemiplegias that he had seen. In Dr. Abrahamson's case the movements were distinctly choreic, arrhythmical, more or less purposeful and of slightly explosive character. They were slower than ordinary chorea minor, and not so fast as electric chorea, yet they were distinctly choreic as opposed to myoclonic and myotonic. It would be interesting to know the part of the nervous system that was responsible. The movements were evidently the result of encephalitic processes and were certainly not hysterical. Dr. Sachs' belief was that it was a ganglionic disturbance near one of the larger ganglions and not a cortical disturbance.

Dr. Michael Osnato stated that he had had the first patient under observation since May, 1920, when he was shown to the students at Columbia as a case of encephalitis. He had noticed a rhythmicity of movement, the movements occurring four or five to the second. A considerable improvement had undoubtedly occurred. Malarial parasites had been detected in the patient's blood and suitable treatment had been given.

Dr. Smith Ely Jelliffe said that the syndromes of postinfluenzal and postlethargic encephalitis were becoming more interesting, and they were turning up in greater abundance in private practice, as the increasing attention given to them, in the advancing masses of the literature, was making physicians more observant of the possibilities. These cases had been in existence for many years but heretofore had been distributed into larger and looser nosologic conceptions; and their etiologic relationships to the infections, especially to that of influenza, were largely overlooked.

In the cases under consideration, particularly the case of Dr. Davis, emphasis had been laid on the segmental distribution of the tonic fixation. It involved an entire limb. One point of possible localization had been omitted thus far in the discussion to which Dr. Jelliffe thought attention might be directed. This was the possibility of cerebellar pathway disturbance in certain types of cases, of which Dr. Abrahamson's case might be an example. The studies of Bolk and Rynberk have cleared up much of our knowledge concerning the localization of function of limbs in the cerebellum, and some cases were on record in which segmental motor disturbances of a choreic or tic-like character, as seen in Dr. Abrahamson's case, could be referred back to foci of destruction in the cerebellar mechanisms. Thus Klein (*Monatschr. f. Psychiat. u. Neurol.*, 1918) reported an interesting choreic-like disturbance from a small cyst in the dentate nucleus. As the fatal cases are usually fulminating and rarely afford opportunity to clear up the complete pathology, it would probably take many years before the valuable localizing lessons could be gained from the study of these residuals of mesencephalic involvement.

Dr. Abrahamson, in summarizing the discussion on his patient, said that he doubted the correctness of the localization in the cerebellum, since the patient showed none of the symptoms of cerebellar disease. His case was distinctly one of disease of the communicating pathways and not of the centers.

A CASE OF PYRAMIDAL SCLEROSIS FOLLOWING ELECTRIC BURNS. Presented by Dr. Foster Kennedy.

The patient had been working in front of a dead cable when a charge of 1,100 volts was sent through it. He remembered nothing of the accident and was unconscious for eleven hours after it. The accident occurred six years ago, and he was paralyzed immediately thereafter. He now has spastic legs and double ankle clonus. He has control of the bladder, but incomplete control of the rectum. A large scar is visible on the left scapula, showing the point of ingress of the charge, and a smaller one is found on the left buttock at the point of exit. There were no neurologic signs of interest except the spastic paraplegia. The burn was a third degree burn. Within two years of the injury the patient began to have a double cataract. The lens in the right eye has been removed. Dr. Kennedy said that his information in regard to nervous system results of this type of injury was slight, and asked for any points of interest from the audience.

Dr. Smith Ely Jelliffe said that by chance he had just finished a revision of the chapter on electric injuries in Peterson and Haynes' "Legal Medicine" and had thus had occasion to review the extensive literature. Results of electric injuries of this type producing myelitis from lumbar foci, were extremely rare, but were well known. A review of the entire literature has been given by Jellinek quite recently.

Dr. Isadore Abrahamson asked whether the pathology of this case would resemble cases of caisson disease.

Dr. Smith Ely Jelliffe pointed out that the force of the current acted as a disruptive agent, whereas the gas in caisson disease had an eruptive effect.

Dr. Michael Osnato was interested to know why the current had selected the pyramidal tracts and had missed the others. It appeared strange that the other tracts and cells and columns of the cord had so completely recovered.

Dr. Smith Ely Jelliffe said that the current was not selective but that now six years after the accident much of the condition had cleared up. Disturbances of vegetative function had existed, however, and were still in evidence, notably the cataract.

CLINICAL SYPHILIS OF THE CENTRAL NERVOUS SYSTEM. Presented by Dr. Junius W. Stephenson.

In a plea for the more fundamental instruction of medical students in the practical aspects of syphilology, a brief classification along lines suitable for complete and rapid instruction was given. In attacking the nervous system, Dr. Stephenson pointed out, syphilis rarely attacks en masse, but produces symptoms referable to a patch of meninges here or there, a blood vessel of small caliber, or a tract in the spinal cord. The accuracy of diagnosis largely depends on the individual's knowledge of the anatomy and physiology of the nervous system. The successful treatment of syphilis accordingly depends on proper discrimination as to what anatomicophysiologic destruction is caused by the initial invasion, the condition of certain structures being unalterable, while others would be amenable to treatment. Thus the importance of the early recognition of syphilis is to be emphasized. When the average patient reaches the neurologist, the damage is, usually, too extensive for much improvement. The ability to recognize the disease in the early stages is therefore of the utmost importance.

The one criterion of syphilis is the Argyll Robertson pupil, but this is not a preliminary sign. Any irregularity of the pupil in patients below the age of 40 years should be considered syphilitic until proved otherwise. A further sign of diagnostic value is one that Dr. Stephenson has styled the "dead man's pupil." Experience is necessary to recognize this sign.

The symptoms of syphilis within the cranium, those of meningitis, isolated gumma, cerebral vascular syphilis, thrombosis of the posterior inferior cerebellar artery, general paresis (meningeal and parenchymatous) and syphilis of the optic nerve, were briefly outlined, following which Dr. Stephenson surveyed the type of syphilis involving the spinal cord, touching on the principle diagnostic points and certain special tests and signs that he has found of value. The entire field of syphilitic disease was rapidly summarized and the earmarks of the various types set forth for rapid and easy assimilation.

Dr. Foster Kennedy, in discussing Dr. Stephenson's paper, spoke of the curious refractive phenomenon which caused what Dr. Stephenson had designated as the "dead man's pupil." Although, as he had said, it required skill in learning to recognize it, yet Dr. Kennedy felt that it had a diagnostic value.

STUTTERING: ETIOLOGY AND THERAPY. Presented by Miss Hannah M. Creasy, by invitation.

Miss Creasy stated that it was her impression that speech specialists should endeavor to gain the cooperation of neurologists if the profession of speech correction were to assume the dignity and gain the respect that it deserved. In her work she had found that there was no royal road to recovery. Her object in presenting her methods was to show the philosophy underlying them.

Stuttering, including stammering, she defined as the inability to speak at will. It is a mental condition, a psychic neurosis, not physical, and frees itself in somatic symptoms, such as cramps and excessive tension of the organs of speech. There is lack of harmonious working between the brain and the speech organs. The speech mechanism is not at fault. It is the mind that is tied, not the tongue. Stuttering must accordingly be traced further back than the symptoms, or even the trauma producing the symptoms.

Every experience which produces a painful effect of fear, anxiety, shame, etc., may act as a psychic trauma, and may produce pathologic results, if it falls on fertile soil. "In order to create nervousness," says Charcot, "two factors are necessary—one permanent (the neuropathic predisposition), the other contingent (the provoking agent)." A shock may have brought out the symptoms, revealing the permanent neurotic background. After the effects of the shock may have been removed and the symptoms cured, another shock may again reveal the constitutional weakness and the cure be undone. This has occurred in many of the so-called shell shock cases in the war.

The dominant quality in the syndrome of stuttering is fear—fear of people, or of speaking before people. On entering school, the stutterer carries his fear over from his home environment to any one in authority, any one he thinks might be hostile and unsympathetic. In the best of environments intelligent sympathy may be lacking. The fear attached to stuttering is a morbid fear. It does not arise from the shock, it arises from an internal condition. The history of frights, shocks, accidents and other attributed causes of stuttering show that they might have resulted in a temporary normal fear. The patient's make-up has predetermined the morbidity of the fear. According to Frink, the manifestation of an anxiety hysteria is a morbid fear and the physical accompaniment thereof. Stuttering may be considered a manifestation of anxiety hysteria.

Accepting the premise that stuttering is a fear neurosis (anxiety hysteria), Miss Creasy's treatment follows along the lines laid down for other psychoneuroses. The work is largely a matter of character building—reorganization of the emotional life. To counteract the attributes of fear, cowardice, and stubbornness, environmental trends are recognized to bring about a more normal reaction to life, to produce fearlessness, self control and moral courage. An example of this method given was that of a student who, when asked why he stuttered more than usual at one time, replied that he had been worsted in a fight. His tendency to expect defeat was pointed out to him and he was told to approach his next fight with his mind made up to win, and to stay at the job until he did so.

The personal equation of the doctor in this branch of therapy, as in all others, is of great importance. The understanding of the patient, and letting him know that the doctor understands his peculiar difficulties, will go far toward altering his attitude toward others. Thus, with the friendly cooperation of teachers and schoolmates, Miss Creasy has found that fear will gradually give way.

The stutterer's bodily health and environment should have careful attention. Segregation of stutterers is not desirable. The symptoms may be removed under institutional care, but the problems in the unfamiliar outside world will cause them to return again. Children should be kept in school and in touch with normal life in every possible way.

To attain fearlessness the patient is given a new method of speech. The sufferer is unable to get over the initial consonant sound to the vowel. The psychic fear causes an overinnervation in the occlusive, consonant sounds, and an inability to coordinate the consonant with the vowel in the syllable. The patient is shown that normal speech differs from his in the weaker consonant obstruction. The work proceeds with the syllable, coordinating the consonants and the vowels. Vowel valuation is made the keynote of the work. The patient is trained to consider the vowels as life savers. The mental attitude of fearlessness and self confidence inculcated by the care of the treatment, together with the scientific aids to phonation, when made clear and reasonable to the stutterer, enabled the work to proceed surely and with lasting success.

DISCUSSION

Dr. A. A. Brill agreed that the genesis of the stuttering was in the situation of the patient. Many cases of stuttering starting after puberty were easy to cure, since the stuttering was a symptom of a neurotic condition. Too much time, he felt, should not be devoted to routine practice. Most of the sufferers should be treated as average psychoneurotics. The prognosis in stuttering is usually bad. The treatment is lengthy and trying. The permanency of the cure cannot be guaranteed.

Dr. Neustaedter congratulated Miss Creasy on her conservatism and perseverance. He remarked that stammerers do not stammer when they sing. If the patient can be kept talking slowly the stammering can be largely overcome.

Dr. Smith Ely Jelliffe agreed with Miss Creasy's final summary of the extreme complexity of the situation. He did not believe that even the majority of cases could be ranged in the anxiety hysteria group. The reasons for the types of results obtained lie in the nature of the beginning of the neurosis. The compulsive type, beginning in the auto-erotic period is treated with extreme difficulty, especially when the muscle erotic is involved. Such patients are prone to relapse and require much time. Miss Creasy's effort in establishing a transference is sane and advisable. The most promising part of her presentation, Dr. Jelliffe felt, was the beginning of cooperation between the layman and neurologist. There are not enough neurologically trained physicians to handle psychoneurotic problems in the community. If intelligent cooperation can be established between the neurologist and the sincere helper, much help can be obtained.

Miss Hannah Creasy, in closing, said that since the work in public schools had been developed, rapid advance had been made. Cooperation and team work made the advance more successful. The work in the schools was more favorable than in private work, since in the home there is backsliding. Until the parents will cooperate, therefore, training in schools is advisable. Team work is most necessary to combat the time required and the difficulty of the cure.

Book Reviews

STUDIES IN NEUROLOGY. By Henry Head, M.D., F.R.S., in Conjunction with W. H. R. Rivers, M.D., F.R.S., Gordon Holmes, M.D., G.M.G., James Sherren, F.R.C.S., Theodore Thompson, M.D., and George Riddoch, M.D. Two volumes. Cloth. Price $17. Pp. 862, with 182 illustrations. New York: Oxford University Press, 1920.

These volumes consist mainly of a number of papers published by Head and his co-workers in *Brain,* over a period of time extending from 1905 to 1918. No material change has been made in their arrangement. Each paper detailed the methods of testing sensation. These pages have been eliminated, and a separate chapter in which they are combined has been added, as have also an introduction and an epilogue dealing with the common aims that underlie the various researches. Finally, in an appendix, some of the most serious criticisms of their work have been seriously considered.

The first volume consists of two parts: the first devoted to an introduction and methods of examination; the second to the peripheral nervous system. The second part consists of three papers: "The Afferent Nervous System from a New Aspect," "The Consequences of Injury to the Peripheral Nerves," and "A Human Experiment in Nerve Division." The profound studies described in these three papers resulted in a new conception of the physiology of sensation, which has been widely accepted. The results of their brilliant studies led Head, Rivers and Sherren to conclude that the sensory mechanism in the peripheral nerves consists of three systems: deep sensibility, protopathic sensibility and epicritic sensibility. Whereas previous observers had stated that sensation is diminished over the full area usually assigned to the injured nerve and lost completely over a small portion only, they have shown that this diminution of sensation is, in reality, a total loss of sensibility to stimulation with absorbent cotton, to the compass test, to the painless interrupted current, and to degrees of temperatures between 22 C. and 40 C. In this area are felt only the stimuli affecting protopathic sensibility, such as the prick of a pin and temperatures below 20 C. and above 40 C.

In the chapter on injury to the peripheral nerves are found some illuminating observations on changes in the skin and nails and comments on paralysis, emphasizing the effect of supplementary motility.

The second volume is devoted to studies of "The Grouping of Afferent Impulses," "Sensory Disturbances from Cerebral Lesions," "Sensation and the Cerebral Cortex," and the automatic bladder, excessive sweating and some other reflex conditions in "Gross Injuries of the Spinal Cord."

The chapter on the grouping of afferent impulses is concerned with observations showing that "as soon as a sensory impulse reaches the first junction in the spinal cord it becomes shunted into tracts devoted to the conduction of impulses, grouped in a way different from that found in the peripheral nerves." It is no longer a question of protopathic, epicritic and deep sensibility; each tract in the central nervous system is devoted to the conduction of one of the specific impulses, such as pain, touch, heat and cold. After showing that afferent impulses that reach the central nervous system by way of the peripheral nerves undergo rearrangement within the spinal cord, the authors attempt to trace their course upward, indicating the relation of each set of impulses to the other. Painful, thermal and tactile impulses ultimately pass from the point of entry to the opposite side of the spinal cord, but before they pass across they have undergone the characteristic recombination that occurs in the same half of the spinal cord by which the impulses enter.

The observations noted in injuries of the spinal cord permit important conclusions relating to diagnosis, prognosis and treatment in such cases.

Continuing their researches to the brain, there are exhaustive studies on grouping of afferent impulses in the brain stem, sensory disturbances from lesions of the optic thalami and the cerebral cortex, and a completion of their conception of the physiology of sensation with a study of the cerebral cortex and sensation.

The observations made during the late war have reawakened an interest in the problems of sensation, particularly in reference to the peripheral nervous system. It may be predicted that as wide an interest will develop relative to the spinal cord, the cerebral cortex and the brain stem. We owe considerable gratitude to Head and his co-workers for assembling their illuminating studies so that one may view in a comprehensive manner their conception of the physiology of sensation.

The case reports are so full and the illustrations so profuse that the volumes will interest not only neurologists, but also other clinicians and physiologists.

EPIDEMIC ENCEPHALITIS (ENCEPHALITIS LETHARGICA).

By Frederick Tilney, M.D., Ph.D., Professor of Neurology, Columbia University, and Hubert S. Howe, A.M., M.D., Instructor in Neurology, Columbia University. Cloth. Price, $3.50. Pp. 252, with 58 illustrations. New York: Paul B. Hoeber, 1920.

Most of the original material forming the basis of this book, including the illustrations, has appeared in two articles in the *Neurological Bulletin*, one by Tilney and Henry Alsop Riley, the other by Howe. In Part 1, based on the former article, twenty cases are related and subdivided into the following groups: (1) lethargic, (2) cataleptic, (3) paralysis agitans type, (4) polio-encephalitic, (5) anterior poliomyelitic, (6) posterior and (9) infantile (neonatorum). There are brief chapters on analysis of symptoms, prognosis and diagnosis, nature of the disease and treatment. In Part 2, based on Howe's cases, a different classification is used, namely: (1) cases with general symptoms indicating involvement of the nervous system, but absent, scant or fleeting localizing signs, (2) meningitic type, (3) cortical type, (4) pyramidal system type, (5) thalamic type, (6) corpus striatum (extrapyramidal type), (7) brain stem type, (8) cerebellar type, (9) spinal type, (10) peripheral nerve type and (11) multiple diffuse lesion type. This part includes also chapters on general subjects, partly including ground covered in Part 1, and numerous case reports. The two articles that form the gist of the book contain well studied and excellent material and the same is true of the book, but the now exceedingly voluminous literature is not adequately sifted, and on account of the rather disjointed construction of the book the reader will have to spend some time in finding the information wanted. However, many exceedingly useful and dependable data may be gathered from it.

We are grateful to the authors for their work, which just now fills a much needed want, but we hope that before long these authors or some one else will produce in monograph form a thorough and all-inclusive critical review of this timely subject.

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